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ABSTRACT

These five papers present a compact summary of current biological, behavioral and psychosocial research on cigarette smoking behavior. They focus especially on the addictive and dependence processes related to smoking and its effects on health. While much is known, recommendations for further research are suggested on topics including: nicotine, withdrawal, behavioral pharmacology, abstinence, peer pressure, and treatment. An extensive reference list is included. (JC)

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The Behavioral Aspects of Smoking

Editor:
Norman A. Krasnegor, Ph.D

NIDA Research Monograph 26
August 1979

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Foreword

The National Institute on Drug Abuse has been assigned a leadership role in developing new knowledge of the behavioral aspects of smoking, particularly as this relates to the addictive and dependence processes associated with cigarette smoking. The reprinting in the NIDA Research Monograph series of "The Behavioral Aspects of Smoking," Part II of the 1979 Report of the Surgeon General on Smoking and Health, is in keeping with that role. The five papers constitute a significant document for behavioral scientists and others with special interest in this field. They provide a compact summary of current biological, behavioral, and psychosocial research on cigarette smoking behavior.

Concern about the damaging effects of this widespread behavior on the public health, generated in part by the 1964 Report on Smoking and Health, led to the preparation of the updated and expanded 1979 Report. NIDA's mandate was to present the current scientific information on the processes of smoking behavior. Four chapters included here are the result of this work. The National Institute of Child Health and Human Development was asked to summarize the literature on cigarette smoking in adolescents; the fifth chapter presents their contribution to this study. In addition, the extensive references which accompany these papers are in themselves a valuable resource. Dr. Norman A. Krasnegor, of the Clinical/Behavioral Branch of NIDA's Division of Research, has added an introduction which offers an overview of the scientific progress to date as well as directions for future research in the behavioral aspects of smoking. Dr. Krasnegor has had a primary role in overseeing NIDA-supported research to understand cigarette smoking behavior and the common processes which underlie dependency.

This monograph is a pertinent addition to NIDA's other publications on smoking research (NIDA Research Monographs 17, Research on Smoking Behavior, and 23, Cigarette Smoking as a Dependence Process) and on behavioral studies of substance abuse, including smoking (NIDA Research Monographs 20, Self-Administration of Abused Substances:

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Methods for Study, and 25, *Behavioral Analysis and Treatment of Substance Abuse*). We hope that this volume will be helpful to the research community and that it will serve as both a basic reference and a stimulus to new studies on cigarette smoking behavior.

William Pollin, M.D.
Director
National Institute on Drug Abuse

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Introduction

Norman A. Krasnegor, Ph.D.

The papers presented in this monograph are representative of the various aspects which are important in studying smoking behavior. The initial chapter by Murray E. Jarvik focuses on the "Biological Influences on Cigarette Smoking." Ovide Pomerleau highlights the mechanisms involved in "Establishment, Maintenance, and Cessation of Smoking." The next two chapters, "Smoking in Children and Adolescents: Psychosocial Determinants and Prevention Strategies," by Richard I. Evans and "Psychosocial Influences on Cigarette Smoking," by Lynn T. Kozlowski, underline the large place that this behavior has in our society. Terry F. Pechacek, in the last chapter, "Modification of Smoking Behavior," reviews the vital question of treatment for changing the behavior. Together these papers provide an excellent reference for the current state of the knowledge on tobacco dependency. They are especially important since, though much is known about the consequences of smoking, a great deal still has to be learned about and from the behavior itself.

Smoking is clearly a question of enormous concern for the public health.[1] Last year 54 million Americans consumed 615 billion cigarettes. The economic and social expenditures for the nation were enormous. Pinney (1) estimates that health costs associated with smoking were $27 billion for 1978. The Surgeon General's Report on Smoking and Health (2) indicates that in that same year, 325,000 premature deaths linked to cigarette smoking occurred. Research on the factors which underlie the initiation, maintenance, and cessation of this behavior is of the highest priority from the public health viewpoint since such knowledge is essential for the development of workable treatment approaches and effective prevention strategies.

This paper provides an overview of cigarette smoking from an applied behavior analysis perspective; reviews what is known concerning withdrawal, relapse, and abstinence; and suggests new directions for research.

INITIATION AND ESTABLISHMENT

The enigma of why people continue to engage in a behavior which has such dire consequences for their well-being is still with us. One useful approach to developing the knowledge base that can elucidate this paradox is that provided by the experimental analysis of behavior. Within this framework, cigarettes are viewed as powerful reinforcers which strengthen and maintain behaviors that lead to their use. While little prospective experimental data exist on how people start to smoke, retrospective and anecdotal observations suggest that peer pressure is necessary for experimentation with and initiation of cigarette smoking. Since smoking is associated with dysphoria during this early phase of the behavior's development, continued use is thought to be dependent upon social support. Once a smoker becomes tolerant to the aversive aspects of inhaled smoke, the positive reinforcing properties of cigarettes predominate, the behavior is established, and the social support provided by peers diminishes in importance (3).

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[1] The remainder of this chapter is adapted from a paper presented by Dr. Krasnegor at the Fourth World Conference on Smoking and Health, Stockholm, Sweden, June 18-21, 1979.

MAINTENANCE

Over time, cigarette smoking comes to be maintained by operant and Pavlovian conditioning mechanisms. Conditioned stimuli (e.g., sight of people smoking, time of day, etc.) both set the occasion for the behavior to occur (operant model) and trigger internal physiological events such as craving and discomfort (Pavlovian model). These antecedents increase the chances that smoking will occur in their presence, and, since such events are themselves so likely to occur, help to insure that the behavior is maintained.

While it has not been definitively established, the choice for the most likely constituent in cigarettes which reinforces smoking behavior is nicotine. There are several lines of evidence which support this assumption.

First, we know that nicotine can be discriminated by experimental animals (4). This implies that the drug has a central nervous system effect, it can alter the affective state of an organism, and such a state dependency may play a role in maintaining the behavior.

Second, we know that nicotine is self-administered intravenously by rats and monkeys (5). This finding means that nicotine is a reinforcer, i.e., it strengthens and maintains behaviors which lead to its availability and ingestion.

Third, smokers appear to regulate their intake of nicotine (6,7). This finding suggests that cigarettes are used particularly by established smokers to maintain what, for them, may be a necessary plasma nicotine level.

Fourth, recent neuropharmacological experiments (8) suggest the existence in rat brain of a specific noncholinergic receptor for nicotine. This finding implies that the central mechanism of action for nicotine's reinforcing properties can be studied directly and its biochemical and neurophysical nature can be determined.

Fifth, we also know that the average one-pack-a-day smoker is estimated to self-administer 70,000 boluses of nicotine per year (9). This surpasses by far the rate of any other known form of substance abuse. The implication of this conclusion is that smoking is an overlearned behavior and is therefore difficult to extinguish.

CESSATION, WITHDRAWAL, AND RELAPSE

While there are many approaches to help people stop smoking (10), and 3 to 4 million Americans are reported to quit on their own annually, the literature indicates that maintained abstinence is difficult to achieve. Of those who do succeed in stopping, 75-80 percent relapse within twelve months (11).

Why is the rate of relapse so high? Part of the answer lies in the withdrawal syndrome that occurs subsequent to cessation. Withdrawal is defined as abnormal physiological and psychological changes which appear after cessation of habitual drug use and gradually disappear over time or when use of the drug is reinstated. Shiffman (12), in his extensive review of the literature on withdrawal from cigarettes, reports a variety of changes in physiological, behavioral, and psychological variables.

Blood pressure and heart rate decrease, while REM sleep time and sleep-like EEG's increase. Weight gain is reported, along with the occurrence of nausea, headache, constipation, diarrhea, and excessive eating. Decrements in vigilance and psychomotor performance have also been demonstrated. In the affective domain, smoking cessation is associated with increases in craving, anxiety, irritability, aggressiveness, and hostility. Severity of the abstinence syndrome has been shown to be related to the sex of the smoker (females apparently have more severe symptoms) and the dosage parameters of the cigarettes used prior to cessation (12).

Withdrawal symptoms begin to appear within hours of stopping and some persist for periods ranging from a few weeks to several years. Such alterations in emotional, physiological, and physical status of abstinent smokers are vitally important because they have been cited by researchers (12) as a reason that smokers relapse. When confronted with such changes subsequent to cessation, smokers report that they cannot tolerate the discomfort. They resort to the highest probability behavior (smoking a cigarette) which in the past has relieved the dysphoria they are experiencing, and they achieve a temporary relief from the symptoms. Within a short time, this avoidance behavior is again reinforced by the smoking of yet another cigarette, and the dependence cycle is reestablished.

NEW DIRECTIONS FOR RESEARCH

While the facts outlined above suggest that there is some information on the behavioral bases of cigarette smoking, much more work remains to be done. During the next 3 to 5 years, much new knowledge will be compiled that will shed light on how smoking gets started, how it is maintained, and why it is so difficult for people to give it up. What is needed to achieve this data base is a multidisciplinary approach which employs methodologies from the biological, behavioral, and social sciences. This strategy will insure the development of a comprehensive and balanced understanding.

Based on the literature and the field of smoking research as it now exists, the following foci are recommended as high-priority areas where study should be initiated.

(1) The Role of Nicotine. At present the evidence is accumulating and strongly suggests that nicotine is necessary for the maintenance of cigarette smoking. Studies of the intravenous self-administration of this drug by animals indicate that it can maintain behavior which serves to make it available. A direction of great importance would be the development of animal models which employ the inhalation route of administration. This is the case because nicotine passes most rapidly into the brain via the lungs, and it may be that the reinforcing efficacy is enhanced when administered via this route.

Studies which explore the central site of action of nicotine and drugs which block its effects are of great interest since such research could help elucidate the neuropharmacological and biochemical bases for the reinforcing effects of the drug. Similarly, such investigations could suggest pharmacological approaches for treating dependence on nicotine.

(2) Withdrawal. As mentioned above, systematic studies of the abstinence syndrome associated with smoking cessation should be undertaken. Many questions need answering. For example, what are the most common symptoms reported? How does withdrawal vary with the number of years one has smoked? How does withdrawal vary with the strength of the cigarettes smoked? Is there a conditioned abstinence syndrome associated with cigarette smoking?

(3) Behavioral Pharmacology of Smoking. While there are some data on the topography of smoking, relatively few experiments have been conducted to determine the rate of puffing, volume of puffing, interpuff interval, etc. Such research should be encouraged, especially as these parameters are related to smoking history, nicotine content, stimulus control, etc. The data obtained could be used directly in techniques designed to treat smoking.

(4) Prolonging Abstinence. While there are many procedures used to achieve cessation, the largest problem to be overcome is how to help people maintain that status. Studies which can determine ways to lengthen the period which people remain abstinent are essential.

(5) Longitudinal Studies of Smokers. While there are some exceptions, the general picture suggests that followup in treatment studies is conducted for up to one year. Yet more recent research data indicate that a minimum of two years is necessary to evaluate treatment efficacy for smoking cessation programs. Researchers should be encouraged to employ long term followup designs when evaluating the success of various treatment modalities.

In addition, longitudinal prospective studies should be carried out to investigate the natural history of spontaneous quitters. We know virtually nothing about the millions of smokers who allegedly stop smoking on their own each year and whether such people are successful at maintaining abstinence.

(6) Peer Pressure. Studies which undertake prospective investigations of peer pressure as this construct relates to cigarette smoking should

be initiated. Both laboratory and field studies should be carried out to determine the contribution of peer pressure to the initiation and maintenance of smoking behavior. Such data are essential to help develop effective prevention strategies.

(7) Objective Methods for Validating Self-Reports. There are many studies in the literature on incidence and prevalence of cigarette use and the evaluation of treatment efficacy. Unfortunately, the analysis and conclusions are often based on self-reports only. While some studies do use significant others to corroborate self-reports, few have employed biological assays to validate such subjective data. Work on biological assays such as analysis of breath for CO content and blood for thiocyanate levels is just getting under way. Such work and the development of other biological assays should be encouraged.

(8) Treatment Research. New and innovative techniques, particularly in the context of well-designed multimodal treatment approaches, should be carried out. Such research must include within the design appropriate control groups, random assignment, objective measures of cigarette use (CO, thiocyanate, etc.) and longitudinal followup.

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Biological Influences on Cigarette Smoking

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Introduction

The present chapter reviews current knowledge concerning the biological, biochemical, and physiological correlates of the smoking habit over the three stages of its development. These are respectively: establishment, maintenance, and cessation of the behavior. While there is overlap in each of these stages, one can conceptually divide the process and evaluate from a biological perspective the metabolism and fate of the major constituents of tobacco, the role of nicotine, dependence liability and tolerance associated with the smoking habit, and its physiological correlates. Recommendations for new research initiatives are included where appropriate throughout the text.

Chemistry and Biochemistry of Tobacco Smoke

Cigarette smoke contains a number of compounds that may act as pharmacological reinforcers and facilitate establishment of the smoking habit. Although it is difficult for a psychopharmacologist to ignore the possibility, indeed the probability or certainty, that the chemical composition of cigarette smoke is of vital importance in explaining smoking behavior, there are behavioral scientists who totally ignore chemistry. They focus instead upon the fact that smoking is initiated by peer pressure, and some have expressed the view that oral and manual satisfaction is all that is necessary to maintain the habit. Although it may be inappropriate to go to the opposite extreme and deny the importance of psychological factors in the establishment of the smoking habit, there is much direct evidence that cigarette smoking necessarily involves tobacco and probably nicotine. Cigarettes made of nontobacco materials such as lettuce or cubebs are not popular. The evidence that nicotine is a vital ingredient is somewhat more circumstantial.

A pack-a-day smoker takes more than 50,000 puffs per year and each puff delivers a rich assortment of chemicals into the lungs and bloodstream. Each puff stamps in the habit a little more and augments the establishment of secondary reinforcers, such as the sight and smell of cigarettes, the lighting procedure, and the milieu and context of a meal with a cup of coffee or a cocktail. It would be surprising if chemical factors were not involved in these pleasurable experiences. It is not surprising that such an overlearned habit surrounded by secondary reinforcers is difficult to extinguish.

The possible candidates for reinforcing pharmacological agents in the establishment of the smoking habit are shown in Tables 1 and 2 (*118*). Although nicotine is the most popular suspect for the reinforcing agent in tobacco, there are other possibilities. Tar and carbon monoxide are the two most likely contenders.

TABLE 1.—Cigarette smoke: gas phase components (μg/cigarette*)

Carbon monoxide	13,400
Carbon dioxide	50,600
Ammonia	80
Hydrogen cyanide (hydrocyanic acid)**	240
Isoprene (2-Me-1,3 butadiene)	582
Acetaldehyde	770
Acrolein (2-propenal)	84
Toluene	108
N-Nitrosodimethylamine	0.08
N-Nitrosomethylethylamine	0.03
Hydrazine	0.03
Nitromethane	0.5
Nitroethane	1.1
Nitrobenzene	25
Acetone	578
Benzene	67

* 85 mm non-filter, blended cigarette (U.S.)

** Gas phase portion only (74 μg/cig. in particulate phase)

SOURCE: Schmeltz, I. (*118*).

TABLE 2.—Cigarette smoke: particulate phase components (μg/cigarette)

TPM* wet	31,500
dry	27,900
FTC**	26,100
Nicotine	1,800
Phenol	86.4
o-Cresol	20.4
m- and *p*-Cresol	49.5
2,4-Dimethylphenol	9.0
p-Ethylphenol	18.2
β-Naphthylamine	0.028
N-Nitrosonornicotine	0.14
Carbazole	1.0
N-Methylcarbazole	0.23
Indole	14
N-Methylindole	0.42
Benz(*a*)anthracene	0.044
Benzo(*a*)pyrene	0.025
Fluorene	0.42
Fluoranthene	0.26
Chrysene	0.04
DDD	1.75
DDT	0.77
4,4′-Dichlorostilbene	1.73

* U.S. cigarette, 85 mm, without filter tip, 1968

** TPM-FTC = TPM-H_2O-nicotine

SOURCE: Schmeltz, I. (*118*).

Carbon Monoxide

After nicotine, the substance in cigarette smoke with the most pronounced acute pharmacological action is carbon monoxide (CO). Cigarette smoke contains 1 to 5 percent CO, or 10,000 to 50,000 parts per million (ppm). Carbon monoxide impairs the oxygen-carrying capacity of the blood and may impair functioning of the nervous system. It appears to pose a threat, both acutely and chronically, to the functioning of those with cardiovascular disease. Indeed, it is thought by some (*128*) that the carbon monoxide in cigarette smoke is partially responsible for the increased risk of myocardial infarction and stroke in cigarette smokers. The combination of nicotine, with its catecholamine releasing properties, and carbon monoxide in the blood of smokers may enhance cardiovascular risk.

Little evidence exists to support the hypothesis that carbon monoxide is the reinforcing agent in establishing the smoking habit, although it may interact with nicotine. Quite possibly carbon monoxide may deter a few smokers from establishing the smoking habit because it may induce headaches which would deter further smoking. Other forms of tobacco (snuff and chewing tobacco) that have been used through the ages do not produce carbon monoxide.

Tar

Tar, the particulate phase of cigarette smoke, is also of importance in the establishment of the smoking habit. The possibility that tar may be reinforcing is not so easily disproved because the tar and nicotine content of cigarettes tend to co-vary. One study in which the tar and nicotine were dissociated and varied (*38*) showed that the number of cigarettes smoked was related to the nicotine content but not to the tar. There were indications that there may be an interaction between tar and nicotine. For example, nicotine strongly influenced strength ratings in the expected direction, while high tar cigarettes were actually perceived as milder than low tar. The results are consistent with the hypothesis that people smoke to obtain nicotine, but it would be important to extend and confirm these findings with a wider range of tar and nicotine content.

Nicotine

Nicotine has been proposed as the primary incentive in smoking (*63*) and may be instrumental in the establishment of the smoking habit. Whether or not it is the only reinforcing agent, it is still the most powerful pharmacological agent in cigarette smoke. Nicotine is rapidly extracted, enters the pulmonary circulation, is pumped to the aorta where it stimulates the aortic and carotid chemoreceptors, and may produce reflex stimulation of the respiratory and cardiovascular centers in the brain stem.

Within one circulation period, one fourth of the inhaled nicotine passes through the brain capillaries and, since it is highly permeable to the blood brain barrier (*99*), passes promptly into the brain. Once in the brain, nicotine stimulates nicotine receptors. It also releases various biogenic amines, including the catecholamines and possibly 5-hydroxytryptamine. It may also stimulate some as yet unidentified receptors. It stimulates the emetic chemoreceptor trigger zone in the medulla and, in novices or in large doses, it causes nausea and vomiting. A variety of hypothalamic and pituitary hormones are stimulated by nicotine (*143*). The effects of nicotine on associative centers in the brain are still unexplored but may be of extreme importance in explaining its use and desirability during initiation of the smoking habit. Studies from a number of laboratories indicate that nicotine can have a facilitating effect upon learning and memory in animals (*84*), and possibly in humans (*2*).

The other three-fourths of the inhaled nicotine is delivered to the rest of the body and acts wherever there are nicotinic sites. Thus it stimulates autonomic ganglia with, for example, activation of the gastrointestinal tract. By the same mechanism, it releases epinephrine from the adrenal gland with all the "fight or flight" reactions that this hormone can produce, including mydriasis, tachycardia, vasoconstriction, bronchiolar dilitation, decrease in gastrointestinal motility (though this is generally successfully overcome by nicotinic ganglionic stimulation), and glycogenolysis. It also produces a rise in free fatty acids in the blood, and it can release catecholamines such as norepinephrine from nerve endings and chromaffin cells through the body. These diffuse physiological changes may contribute to increased arousal and thus be important corollaries in the establishment of the smoking habit.

Much of the evidence for the role of nicotine as the primary reinforcer in cigarette smoke is circumstantial. Smokers prefer cigarettes with nicotine than without (*40*), though they will smoke nicotine-free cigarettes.

Cigarettes with a nicotine content of less than 0.3 mg/cig do not do well on the market but recently have been increasing in popularity. Generally, these are smoked by individuals who are trying to cut down or somehow diminish the harmful effects of smoking. Tobacco-free cigarettes are doomed to oblivion almost from the start. Lettuce cigarettes had a brief vogue in the United States, but the two companies producing the two different brands on the market went bankrupt.

It is important to note that low or no-nicotine cigarettes allow their smokers to go through all the motions of smoking. Lighting, handling, and puffing can be the same as with usual cigarettes, so the opportunity for visual, olfactory, and oral gratification is present. It is the rare smoker, however, who continues to smoke cigarettes lacking nicotine for any length of time when the more popular high nicotine cigarettes are available. The most likely explanation for this preference is that nicotine is reinforcing.

Metabolism and Fate of Tobacco in the Body

There is little data relating metabolism and fate of tobacco to the establishment of the smoking habit in adolescence. Differences, however, have been found in the metabolism of tobacco in adult nonsmokers and smokers. Beckett and Triggs (*8*) administered nicotine to smokers and nonsmokers and measured urinary nicotine content. The nicotine content in urine from smokers (55 to 70 percent) was consistently higher than from nonsmokers (25 to 50 percent). It would be useful to do enzyme studies in a large sample of adolescent and preadolescent subjects to determine whether chemical profiles might help predict who will take up smoking and who will not. Also, if there are biological deterrents to smoking, it would be useful to find them.

Predisposing Factors

Genetic

Relatively little is known about biological factors in the initiation of the smoking habit. Many studies that have implicated biological factors in the initiation of smoking behavior attribute the behavior to a genetic predisposition. Initial twin studies by R. A. Fisher (*33*) led him to hypothesize that genotype was a significant variable in smoking behavior. In his survey of twins from Germany and England, he reported that monozygotic twins were more concordant in their smoking behavior than dizygotic twins.

Eysenck (*30*) has measured personality variables and has concluded that smoking behavior is related to the extroversion-introversion dimensions of personality. Eysenck's theory assumes that differences in these dimensions of personality are for the most part determined by hereditary factors. He presents evidence indicating that monozygotic twins are more alike on these dimensions than dizygotic twins, and that cigarette smoking is associated with the extroversion dimension of personality. These data have in part formed the basis for the common genotype hypothesis. This hypothesis states that tobacco smoking and lung cancer (and in the theory of Eysenck, personality factors) are due to a common genetic mechanism (*76*). Subsequent analysis of twin studies have supported (*18*, *119*) and denied (*113*, *139*) a significant genetic influence on smoking behavior. However, Cederlof, et al. (*19*) recently published an extensive review of the data from the Swedish twin registry and concluded that "the constitutional hypothesis as advanced by Fisher and still supported by a few, has here been tested in twin studies. The results from the Swedish monozygotic twin series speak strongly against this constitutional hypothesis." The Chapter on Mortality in this report contains a more complete discussion of this topic.

In general, studies from which inferences about genetic mechanisms and smoking have been made are subject to many of the pitfalls associated with survey-type research. Studies of twins are among the most popular means of assessing genetic factors (*14*). Unfortunately, the small number of subjects used in twin studies (particularly monozygotic) has limited the inferences that can be made about genetic mechanisms. An additional confounder not controlled in twin studies is the prenatal environment. The prenatal environment for monozygotic twins is likely to be more similar (i.e., twin positions, common circulatory factors, etc.) than for dizygotic twins (*88*). Further progress in this area will depend on more exhaustive and sophisticated methods of analysis.

Endocrinological

The importance of endocrine factors in the establishment of the smoking habit has not been explored. There is abundant evidence that hormonal changes in puberty occur at about the same time that individuals start smoking. Retrospective studies indicate that teenage smokers are more outgoing, self-confident, and rebellious toward established authority than their nonsmoking counterparts.

The acute endocrine changes associated with cigarette smoking are difficult to interpret because of non-specific stress factors which may accompany smoking. Winternitz and Quillen (*149*) measured ACTH and growth hormone levels in nonsmokers after smoking two cigarettes. There was a rapid increase in the plasma levels of both hormones, but the authors were unable to determine if the effect was due to the tobacco smoke or to the stress created by smoking. The subjects developed nausea, became pale, and started sweating. In chronic smokers a sharp rise in plasma cortisol was observed after two cigarettes and was maintained for several hours. Growth hormone levels peaked at 1 hour and fell back to control levels during the second hour of measurement. No significant changes were found in LH, FSH, TRH, and testosterone levels.

One of the most frequently demonstrated endocrine effects of nicotine is the stimulation of vasopressin release from the supraoptic nucleus (*5, 46, 110*). Robinson and his colleagues have shown in humans that nicotine stimulates the release of a neurophysin associated with vasopressin secretion. A second estrogen-stimulated neurophysin was not affected by nicotine treatment.

In a similar study, Hayward and Pavasuthipaisit (*46*) measured plasma vasopressin levels in adult female monkeys after intravenous infusion of nicotine (100 μg/lkg/min). A significant increase in circulating vaspressin levels was measured that could, in part, be abolished by pre-treatment with promethazine and diphenhydramine. The association between endocrinological responses and smoking is not clear, however. That smoking *causes* such responses has been established, but it would be important to determine whether these responses in turn reinforce further smoking.

Acute Effects of Tobacco and Its Constituents Upon Establishment of Smoking

Central Nervous System

It is clear that tobacco has reinforcing properties that motivate its users to continue smoking even when they are aware of the possible health consequences. Nicotine appears to be the chemical in tobacco that is most likely responsible for these effects (*63*). When the nicotine and tar content are varied independently, it is the nicotine content that is correlated with ratings of strength and satisfaction (*39*). Numerous investigators have shown that nicotine will release norepinephrine from postganglionic sympathetic sites, acetylcholine from postganglionic parasympathetic sites, and epinephrine from the adrenal medulla. However, the primary sites of reinforcement appear to be in the central nervous system. Oldendorf (*99*) has demonstrated that nicotine readily crosses the blood-brain barrier. Stolerman, et al. (*127*) administered mecamylamine, a central nicotine antagonist, to smokers and observed an increase in cigarette consumption. This change was presumably an attempt to overcome the blockade. Further, when the peripheral antagonist, pentolinium, was administered, no change in cigarette consumption was noted. These data are supported by animal studies indicating that rats trained to discriminate nicotine from saline do not generalize the response to similar drugs (*116*). In a related study, Hirschhorn and Rosecrans (*51*) reported that mecamylamine abolished an established nicotine discriminative response.

An important central nervous system effect of nicotine is its ability to modulate arousal levels. The cortical EEG has been used by many investigators as an index of changes in arousal processes (*58, 66, 135*). When smokers are deprived of tobacco for short periods of time, there is an increase in lower-frequency and high-amplitude waveforms in their EEG, thus indicating a possible state of "hypoarousal." Interpretation of these studies has proved difficult because adequate control groups were not employed. It is possible that the process of inhaling in a manner that simulates smoking will elicit the same EEG changes as smoking a cigarette.

The study of Kales, et al. (*66*) in some ways tempers this criticism in that it demonstrated differences in sleep patterns between nondeprived and deprived smoking conditions. During deprivation, smokers spent more time in REM sleep than during nondeprived states. This result could also be due to nonspecific stress.

Research has shown that animals may self-administer nicotine. For example, Pradhan and Bowling (*106*) studied the effects of intraperitoneal administration of nicotine on self-stimulation in rats. The baseline rate of self-stimulation varied as a function of electrode placement, current intensities, and time spent lever-pressing. At high baseline levels of self-stimulation, nicotine enhanced the rate of stimulation.

These data are consistent with other studies that demonstrate that drug effects are largely dependent upon baseline levels of self-stimulation. In a somewhat different approach, Yanagita (*153*) has studied the reinforcing properties of nicotine by demonstrating that monkeys will self-administer nicotine on a regular basis when given the opportunity. An earlier study by Deneau and Inoki (*23*) presented similar results.

There are very few studies in which nicotine alone has been administered to man in an attempt to produce reinforcement (*64*, *65*, *80*). Johnston injected himself and other volunteers with nicotine and obtained clear evidence of reinforcement. These unique studies were uncontrolled for suggestion, however. There were three studies in which nicotine was given either by ingestion or intravenously, and in all three, it was incapable of completely suppressing smoking, though it usually had some suppressant effect. Indeed, in the experiment by Kumar, et al. (*75*), there was no discernible effect of a rapid intravenous infusion of 1.17 mg of nicotine. Subjects went on puffing their cigarettes just as they did with an equivalent injection of placebo, and there was no delay in latency to the first puff.

The results are disturbing to proponents of the nicotine hypothesis of smoking. It is clear that the intravenous infusions had no effect on the subsequent puffing of cigarettes, whereas the cigarettes smoked immediately preceding the test session had a marked effect both on latency to the first puff and on the rate and volume of puffing. Perhaps the nicotine delivered to the blood and brain were not equivalent in the two conditions. Perhaps the intravenous dose should have been higher; it might have been swamped by the fact that *ad lib* smoking was allowed during the intravenous administration of nicotine. Clearly more research is needed to clarify these results.

If it could be established that central nervous system effects of smoking were reinforcing, it would be important to study these actions in novices.

Cardiovascular System

Before he takes his first cigarette, the novice is not likely to be aware of his cardiovascular system. The first cigarette, however, may have a very profound effect upon the heart and blood vessels of a nonsmoker. The tachycardia may be perceived either as a pleasant or unpleasant sensation. The cardiovascular changes associated with tobacco intake resemble the effects elicited by nicotine alone. Both sympathetic and parasympathetic ganglia are stimulated by low concentrations of nicotine, and nicotine can have sympathomimetic effects by releasing epinephrine and norepinephrine from chromaffin cells in the adrenal medulla, heart, blood vessels, and skin (*139*,. Increases in heart rate (10 to 25 beats per minute), blood pressure (10 to 20 mm Hg systolic, 5 to 15 mm Hg diastolic) and cardiac output (0.5 $l/min/m^2$) typically occur in both nonsmokers and smokers after smoking one or two cigarettes. In addition, digital blood flow and finger and toe temperature fall (*139, 151*).

The acute cardiovascular responses to tobacco and nicotine have been summarized in the Surgeon General's reports on the health consequences of smoking (*136, 138*). These reports list the following acute changes from smoking: increased (1) heart rate, (2) blood pressure, (3) cardiac output, (4) stroke volume, (5) velocity of contraction of the heart, (6) myocardial contractile force, (7) coronary blood flow, (8) myocardial oxygen consumption, (9) arrhythmia induction, and (10) electrocardiographic changes. These effects are assumed to be due to catecholamine release from the adrenal medulla, chromaffin tissue, or sympathetic nerve endings, and are similar to those obtained by sympathetic stimulation. They are to a considerable extent mediated by sympathetic excitation (*139*). These diverse cardiovascular changes may be a significant component in shifting the arousal continuum toward an optimum level for smokers. However, there are no controlled experiments that definitely rule them in or out as contributors to the reinforcing properties of cigarettes.

Maintenance of the Smoking Habit

The biological factors which can be implicated in the maintenance of smoking have, by no means, been thoroughly investigated. A great deal is known about the *harmful* biological consequences of smoking, but very little about the *beneficial* effects. It is evident that some component or components in tobacco and tobacco smoke must be reinforcing, but these have not been unequivocally identified. As noted earlier, the possible candidates for reinforcing agents can be seen in the two tables (Tables 1 and 2) from Schmeltz and Hoffman (*118*). The leading contender is nicotine because it is clearly a powerful pharmacological substance and is administered in ways consistent with its action as a reinforcer. There are, however, some inconsistencies in the literature. Yanagita (*153*) has reported low levels of nicotine self-administration in monkeys and rats respectively, while Russell, et al. (*111*) report a lack of evidence for self-administration in man, as well as in other animals. The present discussion focuses upon tolerance to tobacco and its constituents, the metabolism and fate of the constituents, and their physiological effects as they relate to the maintenance of the smoking habit.

Tolerance

By definition, tolerance is manifested by a decreasing response to repeated administration of the same dose of a drug, or by the requirement for increasing doses in order to elicit the same response. Martin (*81*), Jaffe and Sharpless (*61*), and others have proposed models which imply that dependence and tolerance are based upon identical mechanisms. It is difficult to think of an example of a drug to which dependence occurs that does not also involve tolerance. On the other hand, tolerance may occur without dependence (e.g., phenothiazine, antihistamines).

Three kinds of tolerance are apt to occur with tobacco use as with other types of drug use: drug dispositional or metabolic tolerance, tissue or pharmacodynamic tolerance, and behavioral tolerance. The first refers to methods that the body uses to eliminate or to deactivate the drug. For most chemicals derived from tobacco, the liver is the organ most heavily responsible for detoxifying or transforming them into inactive and eliminable forms. The kidney is also important, especially for alkaloids whose water solubility varies with the pH of the solution. The second kind of tolerance refers to changes in the ability of receptors to be activated by the drug at its final site of action. The third type refers to the way in which the subject using the drug changes his behavior to adapt to the effects which the drug repeatedly produces.

Of the compounds contained in tobacco and tobacco smoke (*118*), three are of primary biological importance: tar, carbon monoxide, and nicotine. There is evidence that tolerance can develop to the effects of each of these, although their interaction has scarcely been studied. While there is evidence that tolerance may develop to other components such as acetone and phenol, it is unclear how much they contribute to the pharmacological actions of cigarettes.

Nicotine

Stolerman, et al. (*126*) examined the interaction between pairs of injections of nicotine which varied both in dose and in interval. Two measures of spontaneous locomotor activity of rats in a T-maze were taken: rears and entries. After a single treatment with nicotine, acute tolerance developed as indicated by a shift of the dose-response curve. The dose of nicotine required to produce a given decrement in activity was multiplied by a factor of about 2.4 when a delay of 2 hours was taken between the two injections. When the initial dose was varied, it was found that there was an optimal level for producing tolerance. Higher doses were less effective. An explanation for the relative ineffectiveness of the higher doses in producing tolerance is not available. A general debilitating effect of pretreatment with large doses does not seem to explain it, as rats given a saline challenge exhibited normal motor activity. Perhaps the debilitating effects of a large pretreatment dose and a challenge somehow summate.

Carbon Monoxide

Levels of carbon monoxide achieved in the human body following cigarette smoking increase levels of carboxyhemoglobin. These chronically high levels of carboxyhemoglobin found in smokers can induce polycythemia by increasing hemoglobin levels. These compensatory changes enable the smoker to tolerate increased carbon monoxide levels and to cope with the oxygen deficit produced by cigarettes.

Tar

Tar is defined as the total particulate matter (TPM) collected by a Cambridge filter after subtracting moisture and nicotine. The polycyclic aromatic hydrocarbons are generally blamed for a substantial portion of the carcinogenic activity of tar. They are also powerful enzyme inducers and are undoubtedly responsible for much of the tolerance to themselves and a variety of other compounds produced by smoking. The tar content of cigarette smoke for all brands is determined yearly by the Federal Trade Commission which publishes a listing, along with nicotine content. Tar and nicotine tend to co-vary and thus their effects may be confounded. Obviously, tar is obtained in the smoke from pipes and cigars but not from chewing tobacco and snuff. The latter do not deliver pyrolysis products, such as carbon monoxide, and may thus be somewhat safer. Because the hepatic microsomal enzyme formation is induced by a number of carcinogens in the tar fraction of cigarette smoke, including benzopyrene (*96*), smokers are rendered tolerant to both the therapeutic and toxic effects of a wide variety of drugs (*129*). Even the enzymes in platelets are activated (*53*).

The phenomenon of tolerance to the effects of tobacco products has been clearly demonstrated in both humans and animals. As might be expected, most of the emphasis has focused upon nicotine, but carbon monoxide and tar components also play an important role. As with all other drugs, tolerance varies with subjects and functions. Certain invertebrate forms which feed on the tobacco plant have a high genetically determined tolerance. It is reasonable to assume that even in humans some of the variance in response to tobacco is innately determined and may account for some of the high concordance in smoking behavior seen in identical twins. Other forms of tolerance are clearly the result of experience and develop after exposure to tobacco products. Much more research needs to be done to determine the degree of tolerance which develops in different physiological and psychological functions after tobacco use. For example, it is evident that even in heavy smokers of long duration the heart rate speeds up after each cigarette. On the other hand, nausea and vomiting diminish and disappear with continuing moderate use of cigarettes. It would be very informative indeed to know what changes take place at the putative sites of action of nicotine with chronic use. Do nicotinic synapses at ganglia change in the same way as nicotinic synapses in the brain? Do carbon monoxide and tar constituents have any action on these components or on enzyme systems elsewhere in the body? Answers to these questions will enable us to understand better the physiological basis of the smoking habit.

Tolerance to the effects of cigarette smoke was noted in dogs given cigarette smoke via tracheostomy (*44*). At the beginning of the study the smoke was aversive, but with the passage of time, animals exhibited tail wagging and improved cooperation. In a careful study, Stolerman, et al. (*127*) showed the development of both acute and chronic tolerance in rats. Nicotine administered intraperitoneally to experimentally naive rats depressed activity in a Y-shaped runway in a dose-related manner. After a single intraperitoneal dose of nicotine, acute tolerance to the depressant action of a second dose developed with a definite time course. This became maximal after 2 hours and wore off after about 8 hours. Repeated intraperitoneal doses of nicotine (three times daily for 8 days) elicited chronic tolerance which persisted for at least 90 days after the end of regular treatment with the drug. Tolerance was also produced when nicotine was administered in rats' drinking water and through reservoirs implanted subcutaneously. It appears, then, that tolerance to nicotine in rats can develop quickly, may be easily measured, and persists for prolonged periods after withdrawal. In these experiments, rapid withdrawal of nicotine did not produce the signs of illness which morphine withdrawal regularly produced. The existence of prolonged tolerance to nicotine in rats suggests that the same phenomenon might exist in man. If tolerance to the unpleasant effects of nicotine, such as nausea, developed more rapidly and persisted longer, it might facilitate relapse to tobacco use.

Metabolism

Nicotine

The metabolic fate of 1 mg of nicotine base injected intravenously in humans (actually as nicotine hydrogen tartrate) was intensively investigated by Beckett, et al (*7*). They found that smokers excrete nicotine significantly faster than nonsmokers. None of the smokers reported any nausea from the nicotine injections, but this was reported in varying degrees by all nonsmokers. Haines, et al. (*42*) reported that the plasma concentrations of nicotine were actually higher in smokers than in nonsmokers 1 minute after smoking, but these results were confounded by the fact that nonsmokers were instructed to smoke cigarettes. Obviously smokers were able to inhale more effectively than nonsmokers, in part because they had acquired tolerance to the aversive effects of cigarette smoke on the respiratory passages. Indeed, some of the tolerance that smokers show to cigarette smoke may be correlated with diminished function of the respiratory epithelium and possible depression of taste and smell (*70*). The proposition that heavy smokers adjust their plasma nicotine levels is compatible with the observation that regular smokers commonly consume about 20 to 30 cigarettes during the smoking day (approximately one every 30 to 40 minutes) and that the biological half life of nicotine in humans is approximately 20 to 30 minutes (*57*, *111*). While studies with intravenous nicotine (*80*) show changes in smoking rate apparently due to nicotine concentration in the blood, studies using nicotine gum (*73*) did not show the same effects as intravenous nicotine. It is postulated that the nicotine derived from the gum is absorbed in the intestine and sent to the liver directly via the portal and is there metabolized; therefore less nicotine enters the systemic circulation. Most investigations of smoking rates indicate that much more than plasma nicotine level regulation is involved.

Carbon Monoxide

The metabolism of carbon monoxide involves both the exhalation of the substance from the lungs and a compensatory increased hematocrit to increase oxygen capacity. The former is slowed by the high affinity of carbon monoxide for hemoglobin, and the latter's rate is limited by the process of hematopoiesis. Carboxyhemoglobin has a half life in the body of at least 3 to 4 hours (*137*). It is not known whether the metabolism of carbon monoxide plays a physiological role in the maintenance of the smoking habit.

Tar

Some examples of the effects of induction of microsomal enzymes are cited by Hunter and Chasseaud (*54*). Aryl hydrocarbon hydroxylase is regularly induced by smoking. Benzopyrene hydroxylase and aminozao dye N-methylase were higher in the placentae of pregnant smoking women than in those of nonsmokers. Since tar induces the enzymes of its own metabolism, the smokers might be expected to continue to smoke so as to maintain the levels of tar in the blood, thereby maintaining the action of tar on the metabolism of toxic substances, as discussed above. Metabolism of benzodiazepines, propoxyphene, pentazocine and phenacetin is increased in smokers. Xanthines such as theophylline are also metabolized more quickly in smokers (*105*) and, by inference, so should caffeine be metabolized more quickly. Perhaps this is why heavy smokers drink more coffee than nonsmokers (*9*).

Dependence

Dependence may play an extremely important biological role in the maintenance of the smoking habit (*147*). The characterization of tobacco use as a dependence process raises the issue of tobacco withdrawal. Thus, the subject of dependence is deferred to the section on cessation of the smoking habit to be discussed in conjunction with the acute effects of cessation and the abstinence syndrome.

Physiological Effects of Tobacco and Its Constituents in the Maintenance of Smoking

Although a great deal has been written in previous editions of the Surgeon General's Report on the untoward effects of smoking, very little has been said about the factors that might be responsible for the establishment and maintenance of the habit. In the past 15 years the public has been exposed to ample warnings about the dangers of smoking; nonetheless the incidence of smoking remains high. Therefore, it is important to consider both the evidence and hypotheses about why smoking is such a tenacious habit. The actions of cigarette smoke and its components upon the central nervous system, cardiovascular system, and endocrine system might give us a clue to the strength and persistence of the habit.

Central Nervous System

In their study of smokers, deprived smokers, and nonsmokers, Knott and Venables (*72*) showed that the deprived smoker is characterized by a "state of cortical hypo-excitation and that tobacco smoking increased cortical excitation to the level of the nonsmoker." Citing the findings that tobacco smoking improves efficiency, prevents deterioration of reaction time (*35*), and improves learning (*1*, *3*, *17*), they suggest "that individuals smoke to achieve this specific psychological state of increased vigilance and attention associated with alpha frequency."

Nelsen, et al. (*95*) studied the effects of nicotine administered (100 μg/kg) subcutaneously to rats. The rats had electrodes placed in the reticular formation which, when stimulated, blocked visual learning tasks. The nicotine attenuated the electrical stimulation and increased learning. The suggestion is made that the nicotine-induced limbic system activation antagonized the behavioral disruption.

In Carruthers' attempt to isolate the "rewarding centers" (*16*), he used a β-blocker, oxprenolol, to decrease epinephrine and norepinephrine associated with anxiety and smoking. The secondary effects of increased heart rate, blood pressure, and free fatty acids were blocked along with the systemic increase in catecholamines, and yet the satisfaction subjectively evaluated was unchanged. His conclusion was that there may be a hypothalamic norepinephrine release leading to pleasure. It is not clear whether the oxprenolol crosses the blood-brain barrier. The more conservative conclusion would be that heart rate, blood pressure, and free fatty acid increases might not be involved in the pleasure associated with smoking.

In addition to the learning studies mentioned above, recent studies add the following data. Stevens (*124*) studied 115 males on four learning tasks. His conclusion was that those who smoked more than 12 cigarettes per day did significantly less well than the nonsmokers and light smokers. Andersson and Hockey (*2*) showed that, in two groups of 24 female students who were habitual smokers, the group in a control, no-smoking condition showed immediate serial recall equivalent to that of the group allowed to smoke one cigarette. The group not smoking did perform better in incidental memory, such as remembering in which corner the words were presented. This suggested that the cigarette increased attentional selectivity during increased arousal. Elgerot (*28*) used three complex and two simple tests to determine differences between a 15-hour abstaining group and the same group after smoking freely. In the nonsmoking condition, they improved on complex tests but were unchanged with respect to simple tests. The interpretation is based on the performance-arousal curve; "According to the Yerkes-Dodson law, the optimal level for arousal is lower for complex than for simpler tests." The conclusion is that the combination of the task and the cigarette led to an arousal level too great for the complex tests. An alternative hypothesis is that the smokers were under-aroused and that the abstainers were anxious enough, but not too anxious. The second explanation would account for the finding, but it is not consistent with other authors. Elgerot (*28*) cites the following effects in habitual smokers: (1) decreased hand-steadiness (*36*), (2) improved simple and choice reaction times (*93*), (3) improved driving tasks demanding sustained performance (*48*), and (4) impaired short-term memory but favorable effects on consolidation (*1*). Some of these changes in arousal levels and functioning capacities may be of benefit to the smoker and may reinforce maintenance of the smoking habit.

Other effects of smoking on the nervous system may be positively reinforcing. Decreased acetylcholine axonal transport and synthesis in neurons (*49*) may lead to decreased GI motility and augment the sympathetic response in calming digestion. Other investigators have shown no basic differences in the basic taste sensations between smokers and nonsmokers (*83*).

Cardiovascular System

The most commonly reported acute changes in the cardiovascular system are the following: increase in plasma catecholamines (*4*, *78*), increased heart rate (*4*, *5*, *78*), increased blood pressure (*4*, *5*), vasoconstriction (*43*, *94*), and increased carboxyhemoglobin (*4*, *98*). It is conceivable that cardiovascular changes are associated with pleasant emotional experiences, although Carruther's (*16*) β-blocking experiment would not support this possibility. Possibly decreased peripheral blood flow (*43*) is a heat-conserving mechanism which may drive individuals to smoke. The increased viscosity of the blood due to increased hematocrit (*140*) is of unknown benefit on a chronic basis.

Endocrinological System

Although there has been much recent research on endocrine effects of smoking, the role these play in the smoking habit has scarcely been examined. With the development of more refined and more economical techniques for measuring hormones and their actions, we can expect an acceleration of research in this area.

Hayward and Pavasuthipaisit (*46*) administered IV nicotine to monkeys, causing an increase of arginine vasopressin (AVP) without changes in plasma osmolarity. Husain, et al. (*55*) and Robinson (*109*) also demonstrated the release of AVP plus neurophysins in humans.

Cryer, et al. (*22*) demonstrated that growth hormones and cortisol are released by smoking and are unaffected by β-blockers. Both are involved in protein and carbohydrate metabolism. Perhaps their effect on plasma glucose helps reinforce the smoking habit. Similar results were found by others (*100, 141, 149*).

Perhaps a factor involved in maintenance of smoking is the increased lipolysis due to release of catecholamines and glucocorticoids. A common reason given for returning to smoking is weight gain (*150*).

Other endocrinological effects of nicotine include increased gastric HCl secretion (*24, 89*), decreased pancreatic bicarbonates and water secretion secondary to inhibition of secretin (*11, 12, 13, 25*), changes in placental hormones (*21, 122*), alteration in prostaglandin formation (*144*), and delayed LH surge in female rats (*85*). Also, it is known that in smokers there is decreased sperm quality and distribution (*117*). Smokers and nonsmokers do not seem to vary in LH, TSH, T4, and FSH (*149*), however.

Cessation of the Smoking Habit

Early Effects of Cessation

Cessation of smoking is associated with alterations in CNS, cardiovascular, and other physiological functions. Whether these are true "withdrawal" phenomena characterized by a rebound or merely a return to normal levels still remains to be determined. It is evident, however, that significant changes do occur.

A number of physiological changes have been observed on withdrawal from tobacco. Decreases in heart rate and diastolic blood pressure are observed as early as 6 hours after withdrawal (*91*). These changes persist for at least 3 days (*71*), (*146*) and perhaps for 30 (*37*). Decreased excretion of both adrenaline and norepinephrine (*92*) and various metabolic changes have also been observed (*37*).

These metabolic and peripheral effects, which are often associated with decreased arousal, have been supported by EEG studies showing increases in low-frequency activity (*135*) and alterations in cortical alpha frequencies (*72*). Ulett and Itil (*135*) recorded cortical EEG from heavy smokers (one pack of cigarettes per day) in an attempt to detect EEG changes associated with acute withdrawal. Baseline EEG measurements were obtained while the smokers engaged in their normal smoking pattern and were compared with data from the same individuals after they were deprived of tobacco for 24 hours. It was found that there was a significant increase in the low-frequency EEG bands (3-5-7 cycles/sec) during deprivation. This effect was readily reversed after the subjects smoked two cigarettes within a 5-minute period.

In a similar study, Knott and Venables (*72*) did a computer analysis of cortical alpha activity in male nonsmokers, smokers asked to abstain for a 13- to 15-hour period, and smokers who continued their normal pattern of smoking. Analysis of variance of pre-smoking alpha activity indicated the mean alpha frequency of the subjects in the deprived group was significantly lower (9.3 Hz) than in the nonsmoking group (10 Hz) and nondeprived group (9.9 Hz). When the deprived group smoked two cigarettes, the alpha frequency increased to the levels of the nonsmoker and smoker control groups. Thus, there is evidence for a rebound effect and a true withdrawal reaction. The data are interpreted as indicating that deprived smokers are in a state of cortical "hypo-excitation," and that smoking has the effect of increasing excitability to levels comparable to those found in nonsmoking and nondeprived groups. Since all groups were equal on measures of extroversion, the authors hypothesize that they have described a true "smoking factor" rather than a difference due to personality. Alternatively, one could conclude from the same data that the results obtained are due to the removal of an arousal-producing drug from a group of people who are ordinarily hypo-aroused.

Numerous other physiological changes have been noted to occur after cessation of smoking. Ejrup (*27*) reports that weight gain is a common sequela to cessation. Although not generally observed, he reported that, in a number of patients, blisters in the mouth occurred along with constipation upon cessation of smoking. If the patients resumed smoking, the blisters disappeared.

Krumholz, et al. (*74*) have measured changes in cardiopulmonary function at rest and during exercise 3 and 6 weeks after cessation of smoking. All subjects had smoked more than one pack of cigarettes a day for at least 5 years. Changes during exercise were measured on the standard bicycle-ergometer test. Following 3 weeks of abstinence, heart rate, oxygen debt, and ratio of oxygen debt to total increase in oxygen uptake during exercise were significantly reduced. In addition, expiratory peak flow and D_L were significantly increased. Pulmonary compliance increased after 3 weeks and continued to do so at 6 weeks. At 6 weeks, maximum voluntary ventilation and inspiratory reserve volume were increased and functional residual capacity was decreased.

Glauser and colleagues (*37*, *38*) studied seven subjects before and 1 month after cessation of smoking. The following measures were found to have changed significantly: (1) body weight increased from a mean of 188 to 195 pounds, (2) body surface area increased from 2.03 to 2.05 m, (3) heart rate decreased from 60 to 57 beats per minute, (4) sugar levels (30 seconds after eating) fell from 137 to 123 mg percent, (5) protein-bound iodine decreased from 5.1 to 4.6 μg percent, (6) serum calcium decreased from 10.2 to 9.7 mg percent, and (7) oxygen consumption decreased from 283 to 260 ml of oxygen/min. The authors concluded that the metabolic change that follows cessation of smoking may be one important variable that causes an increase in weight.

Myrsten, et al. (*93*) have studied chronic smokers who smoked for 5 days, abstained for 5 days, and smoked for 5 additional days. Results from this group were compared with those from a nonabstaining group of smokers. A number of physiological differences were noted during the abstinence period. Adrenaline and noradrenaline excretion levels decreased, skin temperature increased, heart rate decreased, and hand steadiness improved.

Accompanying these objective changes in physiology and performance are subjectively reported changes in physical symptoms, arousal, and mood. These have been reported in studies of smokers sampled while actually undergoing withdrawal (*34*, *41*, *146*), as well as in retrospective studies of ex-smokers up to 14 years after cessation (*15*, *34*, *82*, *103*, *112*, *131*, *152*). Although the specific symptoms reported in each study differ, as does the percentage of abstinent smokers reporting each symptom, a consistent pattern of symptoms can still be discerned. Common among the physical symptoms reported are nausea, headache, constipation, diarrhea, and increased appetite (*41*, *92*, *146*). Also reported are disturbances of arousal, including drowsiness and fatigue, as well as insomnia and other sleep disturbances (*92*, *152*). Inability to concentrate is a common complaint and is consistent with objective assessments of the concentration of smokers in abstinence (*46*). Thus, the objective changes reviewed above appear to be reflected in the subjective experience and self-reports of deprived smokers.

Long Term Effects of Cessation

Once a smoker gets past the initial 3- to 14-day withdrawal effects (*45*, *59*, *120*), what biological factors tend to encourage the now ex-smoker to continue abstinence? The factors opposing most ex-smokers' attempts to refrain seem to win out, since relapse is so frequent. In all cessation methods described, about two-thirds are able to attain some degree of abstinence for a short duration, but about half of these return to smoking in 1 to 2 years (*20*, *68*). Is it the methodology of cessation or the post-cessation factors which determine continuation of abstinence? Kasl (*69*) claims "there is evidence that smokers who stop spontaneously have a lower rate of relapse than those who seek help and participate in some sort of program." The effects of cessation on the central nervous system, cardiovascular system, and endocrine system which might encourage continued abstinence will be discussed along with some of the psychobehavioral components.

Cardiovascular System

When a smoker terminates his intake of tobacco, he reduces his risk in a number of cardiovascular diseases: coronary heart disease (*29, 50, 67, 123*), cerebrovascular accidents (*50*), recurrence of myocardial infarction (*29*), sudden death from CHD (*67, 123*), myocardial infarction (*123*), and complications of atherosclerosis (*101*). These reduced risks are measurable on populations, but what cardiovascular benefits of cessation exist to individuals? One report says that the subendothelial edema of small arterioles and vasa vasorum is secondary to the carbon monoxide of cigarettes and that this, including coronary arteries (*5*), tends to return to normal after 5 to 10 years of cessation. This might reinforce cessation, especially in ex-smokers with angina pectoris or other ischemic heart disease. Janzon (*62*), using venous occlusion plethysmography on the calf, found that after 8 to 9 weeks of cessation peripheral blood flow increased measurably, whereas the control group of continuing smokers actually decreased their peripheral blood flow. It is likely that this improvement of circulation would be accompanied by a sense of well-being and reinforce abstinence as time progressed. The decrease in heart rate and blood pressure (*52*), along with decreased catecholamines, may be a factor in continuing abstinence. Related to the cardiovascular benefits of cessation, it was found that peak-expiratory flow rates of 57 liters/min resulted (*90*), an increase which would be positively reinforcing, especially in active ex-smokers.

Endocrinological System

If the metabolic rate declines (*52*), the major effect would be increased weight, as has been noted by many (*34, 37, 82, 148*). This would tend to reinforce smoking in most people. But there may be some unseen benefit of decreased metabolism in those who are either able to maintain their weight or who are not self-conscious of weight gain.

In Pearson's study of theophylline metabolism (*102*), he found that smokers' half-life of theophylline was 4.2 hours while nonsmokers' was 7.1. Upon cessation, the normalization (toward 7.1) took 3 months to 2 years, implying that there may be induced enzymes in the smoker which do not readily normalize. This may be indicative of other metabolite-clearing processes and, because the normalization effect is gradual, may keep the ex-smoker in a "smoking" state so that he does not "miss" this aspect of smoking. Is it possible that this kind of normalization is responsible for so many returning to smoking after 1 to 2 years (*20, 68*)? Another possible influence may be in sex hormonal levels. After 3 months there is improved quality of sperm motility and density as well as fertility (*117*).

Other Effects

Pederson and Lefcoe (*103*) used the Jackson Personality Inventory and a modification of the Reid-Ware Internal-External Control Scale and found no difference between smokers and successful ex-smokers. They point out that ex-smokers have usually tried to stop at least once and failed, have stopped for health reasons, have experienced cravings and discomfort, and have used substitutes. The fact that spontaneous quitters are more successful than those who get help (*69*) implies that they are either more strong-willed and independent, primed to give up the habit because of other negative factors, or less dependent upon cigarettes. West's description (*145*) of ex-smokers is that they are more likely to be male, older, have smoked less before cessation, started smoking at a later age, have a milieu that is supportive of their stopping, and have fewer indices of neurosis and few psychosomatic symptoms. Lebowitz and Burrows (*77*) discuss the finding that ex-smokers have higher incidence of diagnosed disease and less incidence of symptoms when compared to smokers, suggesting that when it "becomes official" that smoking caused an illness, the smoker will quit more readily than if his symptoms are unattached to etiology or specific pathology.

Another possible effect of cessation may be decreased "chest pain" in those having gastroesophageal reflex, as discussed by Bennett (*10*).

By far the the most common, and clinically the most important, symptom to appear following withdrawal from tobacco is craving for tobacco. The best estimates indicate that 90 percent of all smokers in withdrawal will verbalize their need for cigarettes (*41*). Moreover, among smokers who have been abstinent for 5 to 9 years, one out of five report that they continue to have at least an occasional craving for tobacco (*34*). The importance of craving lies not in its universality or persistence, but in its relation to the clinical goal of modifying smoking behavior. Indeed, the importance of the tobacco withdrawal syndrome in its entirety is based on its provocative role in causing relapse among abstinent smokers.

Dependence

As stated earlier, characterizing tobacco use as a dependence process necessarily raises the issue of tobacco withdrawal. Some authorities believe an abstinence syndrome is crucial to the definition of drug dependence. Indeed, some of the initial reluctance to label tobacco as a dependence-producing substance rested on doubts concerning the existence of a tobacco withdrawal syndrome. This was the position taken by the Surgeon General in 1964, when first alerting the country to the dangers of tobacco. Since then, there has been an accumulation of studies which suggest that withdrawal from tobacco does produce a variety of signs and symptoms which can be characterized as a *tobacco withdrawal syndrome*. Although the syndrome is variable and is only roughly described and understood, its existence is no longer a matter of great controversy. It is characteristic of withdrawal syndromes that their severity is dose-dependent (*60*). Therefore, it is expected that heavy smokers would report more severe withdrawal symptoms than light smokers.

The inconsistency of the effect of deprivation is reflected in the literature. Studies by Myrsten, et al. (*92*) and Mausner (*83*) report no differences in this regard between light and heavy smokers. In contrast, Burns (*15*) reports that subjects who suffered withdrawal symptoms had smoked an average of 6.9 cigarettes/day more than asymptomatic subjects ($p < .01$). Wynder, et al. (*152*) report that the proportion of abstinent smokers reporting more than one withdrawal symptom increases with baseline consumption.

Another possible confounding factor is that, because smokers can vary their smoking consumption in other ways—depth of inhalation, number of puffs, etc.—cigarette consumption may actually be a very poor measure of dose. Also, differences in nicotine metabolism introduce variability in dose even among those who consume similar amounts of nicotine. Thus, estimating a smoker's dose may require measuring serum levels of nicotine or its metabolites. In the one study which has approached this problem, Zeidenberg, et al. (*154*) found among men a higher and significant correlation between serum cotinine levels before treatment and self-reported "degree of difficulty" in smoking cessation. There is some indication that the severity of the abstinence syndrome is dose-dependent, but much ambiguity remains. Because dose dependency is so characteristic of withdrawal syndromes from other substances, establishing this effect for tobacco would be an important step toward an understanding of tobacco dependency. Further research into the relationship should probably proceed along the lines followed by Zeidenberg, et al., using serum cotinine levels rather than cigarette consumption as the independent variable. Dependent measures should include more refined instruments than Zeidenberg and his coworkers' estimates of "difficulty" and should explore both the number of withdrawal symptoms and their severity.

Two studies have focused upon the diurnal variations in withdrawal symptoms (*79, 87*). Data from a study by Meade and Wald (*87*) show that craving in abstinent smokers and in "ad lib" smoking have the same diurnal pattern; that is, the lowest peak occurs when the subject wakes up, gradually rising to a peak in the evening, then falling again at bedtime. Thus, there is a consistent function which describes three different stages of the habit and its control (unrestricted smoking, abstinence, and relapse). The meaning of the underlying function has not been determined. Two different types of explanation are plausible. One focuses on diurnal variation in the internal environment of the smoker, suggesting the influence of some metabolic factor with diurnal variation. The other explanation focuses on the diurnal variation in the social environment, e.g., the timing of work, meals, social contact, recreation, and so on, which affects craving for tobacco. Research which accurately measures craving and relates it to environmental stimulus events and circadian variations in the internal environment could help to decide between these explanations. A more comprehensive understanding of how craving varies with stimulus events and with time of day might prove helpful in designing interventions which help prepare smokers to cope with their craving.

Time Course and Duration

While the time course of the abstinence syndrome following abrupt withdrawal from other dependence-producing substances has been systematically studied (*60*), assessment of the course of the tobacco withdrawal syndrome is made difficult by the subtlety and variability of the symptoms (*139*).

The onset of the syndrome appears to be rapid. Changes in mood (*115*) and performance (*93*) are evident. Early effects are not easily distinguishable from the absence of nicotine effects or the effects of simple frustration. Another study reports data suggesting a decrease in symptoms over time (*41*).

After a marked decline in the first week, the tobacco withdrawal syndrome becomes increasingly less yielding. Estimates of the tobacco withdrawal syndrome's duration have been made in retrospective studies which ask ex-smokers to recall how long their discomfort or "difficulty" lasted. However, these studies produce contradictory findings. Burns (*15*) reports a range from 1 to 12 weeks, and Wynder, et al. (*152*) report that most symptoms were gone after 4 weeks. In contrast, Mausner (*83*) reports that, of the ex-smokers who ventured an estimate, fully two-thirds stated that their difficulty had lasted between 1 month and 5 years. In another retrospective study, 21 percent of the sample of ex-smokers reported at least intermittent craving for cigarettes 5 to 9 years after cessation (*34*). Thus, the duration of the tobacco withdrawal syndrome appears to be extremely variable, and no definitive estimate is yet available.

Degree of Deprivation

Even with continued use, reduction in the dose of a dependence-producing substance typically results in the emergence of a withdrawal syndrome (*60*). It has been shown that smokers who changed to low-nicotine cigarettes often report the gamut of acute withdrawal symptoms described above (*32, 114*). Abrupt and total withdrawal from tobacco, however, is associated with a withdrawal syndrome that subsides more quickly and is no worse than that seen in partial abstinence.

Gradual Reduction and Chronic Withdrawal

Despite the usefulness of gradual withdrawal in other dependency disorders, and despite the congruence of this method with sound behavioral principles, there is considerable evidence suggesting that gradual withdrawal from tobacco is associated with treatment failure (*26, 41, 82, 138*). This discrepancy may be explained by the observation that partial abstinence from smoking leads to *more*, rather than less, discomfort in withdrawal. The result is that a partially abstinent smoker is in a chronic state of withdrawal. Typically, this chronic state of withdrawal leads to relapse and a return to baseline rates of smoking (*26*).

Although this explanation is plausible and fits the data available, it must be treated with caution pending further research. Since all of the research relies on smokers who have chosen whether to quit "cold turkey" or by gradual reduction, there is still the possibility that smokers in some way predisposed to experience a protracted withdrawal syndrome disproportionately choose the gradual reduction method. What is needed is experimental research in which smokers are randomly assigned to "cold turkey" or gradual reduction groups and in which the effects on the course of the abstinence syndrome are evaluated.

Another direction for new research might be to determine the threshold for the onset of the abstinence syndrome in gradual reduction. Perhaps there is some rate or degree of reduction which would not precipitate withdrawal, so that a smoker *could* be weaned from tobacco. In addition to a "rate of reduction" parameter, the onset of severe withdrawal may also be controlled by the absolute dose as well. The relationship between degree of tobacco deprivation and the emergence of withdrawal symptoms deserves further study.

Other Factors Possibly Affecting the Abstinence Syndrome

In addition to the factors already cited, the tobacco withdrawal syndrome may be affected by a number of other variables whose influence remains to be determined. One could speculate, for example, about differences between types of smokers in the severity, pattern, and course of abstinence. A study by Ikard and Tomkins (*56*) suggests that "addictive smokers" experience more severe craving. The smokers in this study were deprived of tobacco only for three hours, however, so that the effects of this typology on the clinical abstinence syndrome are still essentially unknown and deserving of study. Other individual difference variables also deserve study. For example, smoking history, especially such variables as previous attempts to quit and the reason for failure, may affect the withdrawal syndrome. Since the symptoms of withdrawal are relatively ill-defined, the smoker's expectations and set are probably related to his experience of abstinence, as is his motivation to quit (*6*).

Another major factor whose relationship is potentially important, but unexpected, is sex. There is fragmentary evidence suggesting that the abstinence syndrome is more severe in women than in men. Unfortunately, relevant data are too seldom analyzed for this sex difference. For example, Guilford (*41*) reports data separately by sex, but does not submit it to statistical analysis of the sex difference. Yet, of 18 major symptoms reported by her subjects in the first 4 days of abstinence, 15 show some sex difference. Among these 15 symptoms, 13 are more frequently reported by women. The difference is statistically significant (sign test, $N = 15$, $r \leq 2$, $p < .005$). Data reported in a number of other studies line up in the same direction, though the effect fails to reach significance in the individual studies (*104*, *131*, *152*).

It seems likely, then, that women report more abstinence symptoms than men. The importance of this finding lies in its possible relation to another sex difference in smoking cessation: it is well established that women are more likely to fail in smoking cessation efforts. Guilford (*41*), for example, has presented data suggesting that the relationship between withdrawal symptoms and failure in smoking cessation is stronger for women than for men. Thus, women experience more discomfort in withdrawal and are more affected by it in their attempts to quit smoking. It seems likely that this is at least partly responsible for their lower rates of successful cessation.

Nor are organismic variables the only variables relevant here. The method used to achieve cessation may well have an effect on the subsequent withdrawal syndrome. Environmental factors, such as the smoker's social environment, are potentially powerful determinants of the smoker's experience of withdrawal. These and other events, such as social drinking, may produce conditioned craving and are to be considered high risk situations for relapse (*79*). Thus, in addition to the few factors whose influence on the tobacco withdrawal syndrome is known, there are many other potentially important variables whose effects remain to be determined.

Techniques for Measuring Tobacco Usage

The question of how to measure the use of cigarettes is an important one when evaluating the various methods of cessation and the benefits of cessation versus the risks of continuance, and when determining the validity of the reports of study subjects' compliance. (It may also be important in "quantifying" risk factors for disease in current smokers, such as type of cigarette, inhaling pattern, and so forth.) There are five potential sources of information to determine whether or not a person has smoked: urine, blood, breath, saliva, and verbal.

Urine

In the urine, one can assay for the constituents of the cigarette smoke itself or for excretion products that are associated with the physiological effects. Using the Goldbaun and Womanski method, Prado and associates (*107*) measured nicotine excretion in smokers averaging 20 cigarettes/day and found nicotine in the urine in concentrations varying directly with number of cigarettes and inversely with pH of the urine. When deprived of cigarettes for 12 hours, there was no nicotine found in the urine. Trojnar (*133*) compared the urine quantities of adrenaline, norepinephrine, vanilinomandelic acid (a derivative of epinephrine and norepinephrine via monoamine oxidase and catecholamine-o-methyl transferase), and 5-hydroxyindolacetic acid in nonsmokers and those who had quit for at least 6 months. The nonsmokers' and quitters' levels were indistinguishable until the ex-smokers smoked an average of 14 cigarettes. Urine metabolite levels, with the exception of norepinephrine, rose when measured on the second day, (EPI 2.04 g/day, VMA 1.31 g/day, SHIAA 2.4 g/day). In a second study, Trojnar (*132*) found that all four values were increased in smokers over nonsmokers without any discontinuance.

A potential problem in measuring the physiological metabolites associated with smoking is in false positives. This can occur when a subject may have experienced severe anxiety, with increased catecholamines, but did not smoke. The urine nicotine level would seem to be more specific, but both methods would have to be used every 12 hours or less to be accurate.

Blood

One constituent found in blood is carbon monoxide, combined to form carboxyhemoglobin (COHb). Sillett, et al. (*121*) describe the simplicity of using the I.L. 182 CO-Oximeter and the potential for giving subjects quick feedback on their performance. They also say it is possible to detect when those who switch from cigarettes to cigars continue to inhale. Turner (*134*) points out that the average nonsmoker's blood in London has 1.3 percent COHb and that 2 percent is used as a suggestion that smoking has resumed. As cities vary in CO in the air, standards would have to be set depending on locale. When Ohlin, et al. (*97*) confronted 32 patients at an antismoking clinic with their elevated COHb levels, 13 immediately changed their report, admitting recidivism. When considering COHb, one must take environmental and occupation sources of CO into account. Although COHb increases proportionally with number of cigarettes (*125*) and varies with nicotine content (*111*), discretion is necessary in using data.

Serum cotinine levels may be a reliable tool in determining cessation, according to Zeidenberg, et al. (*154*). With a half-life of 30 hours, as opposed to nicotine's 30 minutes, and the relative constancy of the cotinine levels in regular smokers, it is possible in this way to evaluate long-range abstinence.

Breath

The determination of mean alveolar CO partial pressure described by Rawbone, et al. (*108*) makes it possible to determine the carboxyhemoglobin levels of the blood with a correlation of $r=.96$. Also, by subtracting expired CO from inspired, it is possible to determine if a smoker is an inhaler. Vogt, et al. (*142*) used expired CO and serum thiocyanate to assess exposure to cigarettes. Smokers had higher levels of both (CO 8 ppm, SCN-100 μmol/l)—three times greater in those smoking more than a pack a day than in nonsmokers. The correlation between smoking and each variable separately was less than the two combined (CO = .476; SCN = .479; both = .571). The researchers were 99 percent accurate in separating "typical" smoking habits from nonsmokers' habits and hypothesized the possibility of grading intermediate levels for exposure to smoke. No mention was made of environmental or occupational sources of CO or CN.

Saliva

The presence of nicotine in saliva can be determined by gas chromatography and an alkali flame ionization detector (i.e., nitrogen detector) (*31*), but it is difficult to distinguish a pattern of smoking. Nonsmokers separated from smokers can be distinguished from nonsmokers who smoke passively. While this is a sensitive method of measurement, the presence of nicotine in saliva does not prove direct use of tobacco. Using this method, it may be possible to determine a maximal level attainable by passive smoking and use that value as a cut-off in determining probable usage.

Tenovuo and Maekinen (*130*) measured thiocyanate and ionizable iodine in saliva with the following results:

	Thiocyanate (mg/liter)	
	Males	Females
Smokers	210 ± 75	124 ± 46

Nonsmokers	91±44	62±32

	Ionizable Iodine	
	Males	Females
Smokers	7.2±.9	10.1±3.6
Nonsmokers	13.4±9.7	13.9±8.0

Although controls using the same subjects, both smoking and abstaining, were not employed, this technique can adequately separate the values of smokers' and nonsmokers' thiocyanate, especially for males. It should be noted, however, that the overlap between smokers and nonsmokers is considerable and that Vogt found no correlation between the tar content of cigarettes and the thiocyanate levels in saliva.

Verbal

Although there are several biological assays measuring use of cigarettes, McMahan, et al. (*86*) propose using the verbal report of the subject, confirmed by an appropriate associate of the subject. They point out that the correlation between reports of the subject and the associate about the subject's smoking behavior is $r = .86$. While the correlation indicating that the subject and associate agree is encouraging, that may be all this study says. A smoker who does not want the researcher to know his smoking habit accurately will probably either not allow the associate to see him in his true habit or will encourage the associate to "interpret" his smoking pattern along the lines he wishes to portray. Other methods may be used, such as a lie detector, but unfortunately they are beatable.

The only "fool-proof" method of determining use is to observe the subject at all times. Even here the degree of inhalation cannot be accurately determined. Since this approach is highly impractical, biological tests must be employed, and understanding of the potential source of inaccuracy must be considered before drawing firm conclusions. Based on the above descriptions, it would seem that the most practical method would be measurement of nicotine, cotinine, and thiocyanate in the urine. If none of these is found in the urine, the conclusion is that the subject has not smoked (or has borrowed urine). If some nicotine is found in the urine, could it have been from passive smoking? One should note, too, that quantitative analysis of nicotine in body fluids will take on increasing significance, since tar and nicotine levels are being decreased in cigarettes, and researchers will need to know not only whether a subject smoked, but how much.

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Behavioral Factors in the Establishment, Maintenance, and Cessation of Smoking

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Introduction

Smoking is a behavior—a highly complex act which is accompanied by certain cognitions and hedonic states and based on various biochemical and physiological processes. In that sense, research on smoking behavior is at the interface between psychosocial and biological investigations of smoking. While behavioral research has contributed greatly to the technology of smoking cessation, relatively few behavioral investigations have been carried out to elucidate the mechanisms underlying smoking. Because of this, the present chapter will focus on social learning theory and nicotine regulation as general considerations to provide a context for a behavioral analysis of smoking. An evaluation of the contributions from the experimental analysis of behavior to the treatment of cigarette smoking and recommendations for further research will be made. Behavioral research findings on the establishment, maintenance, and cessation of smoking will be summarized. Emphasis will be on those stages (*16*) of smoking which follow initiation and during which the processes that contribute to the tenacity of the habit and its resistance to change are set in motion.

The Social Learning Model

Social learning theory has functioned less as a formal explanatory model of smoking and more as a methodological approach with an associated intervention technology (*35*). The impetus for using behavior modification techniques has been provided by the belief that research procedures which operationalize definitions, emphasize well-controlled empirical research, and are derived from concepts from the experimental laboratory will provide valuable practical and theoretical knowledge—a belief justified by the previous contributions of the behavioral approach toward the understanding of other difficult problems in human behavior. Behavior modification is derived from basic research on animal learning by Pavlov and Skinner. It emphasizes the control of antecedent and consequent environmental events (stimuli) in determining behavior (*4*). Social learning theory represents an extension of behavior modification to situations which involve interpersonal activity, but it incorporates the added explanatory concept of modeling, based on imitation and social reinforcement.

In brief, a social learning explanation of smoking proceeds along the following general lines (*35*): The habit is acquired under conditions of social reinforcement, typically those of peer pressure. Initially the inhalation of smoke is aversive, but after sufficient practice, habituation (or tolerance) occurs, and the behavior begins to produce sufficient positive reinforcement in its own right to be sustained independently of social reinforcement. Smoking now generalizes to situations other than the one in which it was originally acquired. It is important to note that, from the perspective of social learning theory, smoking is seen as a learned behavior from the onset.

The analysis continues as follows: Discriminations between situations in which smoking is punished socially and those in which it is either ignored or favorably received are formed, and various circumstances (both external and internal) begin to control smoking. Insofar as they are associated with smoking, some situations, such as an empty cigarette pack or an annoying telephone call, may serve as conditional stimuli (CS's) which elicit covert responses. These responses (i.e., physiological changes or discomfort, perceived as craving) increase the likelihood of smoking. In turn, they can serve as discriminative stimuli (SD's), setting the occasion for the reinforcement provided by smoking. Moreover, stimuli which are preparatory to the act of smoking, such as the sight of a cigarette, can function as secondary reinforcers for behaviors preceding them (for example, purchasing a full cigarette pack). These cues can also serve as discriminative stimuli for behaviors which follow them, such as lighting the cigarette, thus forming a linked chain of responses (a smoking ritual). For successful termination of the overt act of smoking to occur, the extinction of most or all of the conditional stimuli, secondary reinforcers, and discriminative stimuli which make up the habit is required. The way in which these ideas have been put to specific use in therapy will be discussed in some detail later in this chapter.

The number of emotional events which can influence smoking are potentially quite great. If smoking is seen, in part, as an avoidance/escape response to aversive withdrawal states, then, hypothetically, by a process of stimulus generalization, other dysphoric states (for example, anger, tension, boredom) might also serve as discriminative stimuli for smoking. Also, response generalization may occur. In this case, the smoking ritual serves as a temporary escape (coping response) from various aversive situations (that is, smoking as a response which provides relief). Smoking can be seen, therefore, as a generalized primary and secondary reinforcer providing both positive and negative reinforcement over a remarkably wide array of life situations.

From a social learning theory perspective, smoking is difficult to modify because of its ability to provide immediate reinforcement—nicotine from an inhaled cigarette reaches the brain in seven seconds (twice as fast as intravenous administration from the arm). Furthermore, the habit is tremendously overlearned: at ten puffs per cigarette, the pack-a-day smoker gets more than 70,000 nicotine "shots" in a year—a frequency which is unmatched by any other form of drug taking (*40*). While most smokers recognize that sustained smoking can lead to a variety of unpleasant events, ranging from bronchitis to lung cancer, the ultimate aversive consequences of smoking—though potentially of great magnitude—are delayed and therefore have less influence over ongoing smoking behavior than immediate consequences. This is a situation common to a number of self-management problems (*37*). Unlike alcohol and many other drugs of dependence, there are few immediately noticeable negative consequences (*40*).

To a large extent, behavioral researchers have *assumed* relationships between environmental events and smoking. Treatment practices have been based on general theory rather than on research or a functional analysis of smoking behavior as such. Thus, though part of the promise of social learning theory has been fulfilled, and behavioral concepts may have generated new standards of effectiveness in the treatment of smoking, there has not been a comparable contribution to the understanding of smoking per se.

The Nicotine Addiction Model

A physiologically based model of smoking, emphasizing the key role of nicotine as a reinforcer, has evolved from the work of Schachter (*42*, *43*) and others like Jarvik (*19*) and Russell (*40*). The main focus is on explaining the maintenance of the smoking habit following acquisition. Under this formulation, smoking is viewed as an escape/avoidance response to aversive stimulation provided by periodic nicotine withdrawal in the addicted smoker. An internal regulatory mechanism is implied which detects the level of nicotine and maintains it within characteristic upper and lower limits by regulating the frequency of smoking (and possibly other intake parameters).

Much of the evidence in support of smoking as negatively reinforced behavior comes from a series of innovative experiments conducted by Schachter and his associates over a 10-year span. In one study, Nesbitt (*30*) used the amount of shock a subject was willing to tolerate as a behavioral measure of anxiety. They found that heavy smokers tolerated a higher shock intensity (were less "anxious") when allowed to smoke than when not allowed to smoke; nonsmokers tolerated an intermediate shock intensity. The design did not allow a differentiation between the possibility that smokers tolerated higher shock intensity because of a "sedative" effect of smoking (positive reinforcement) or because smoking constituted escape from withdrawal symptoms perceived as "anxiety" (negative reinforcement). To test for this, Silverstein (*46*) varied the amount of nicotine in cigarettes given prior to shock presentation. He found that smokers given a high-nicotine cigarette tolerated more shock than smokers given low-nicotine cigarettes and that there was no significant difference between smokers given low-nicotine cigarettes and deprived smokers. He concluded that the sensory-motor and oral positive reinforcement provided by low-nicotine cigarettes played a negligible role in increasing shock tolerance compared with the negative reinforcement provided by escape from withdrawal symptoms using high-nicotine cigarettes. Further support came from the observation that nonsmokers exhibited higher endurance thresholds (lower "anxiety") than deprived or low-nicotine smokers. This suggests that "smoking doesn't reduce anxiety or calm the nerves [but rather that] not smoking increases anxiety by throwing the smoker into withdrawal" (*54*). Thus, a nicotine deficit seems to exacerbate the distress induced by aversive shock. Heimstra, et al. (*15*) found the same effect for psychomotor performance on a simulated driving test.

The next problem was to account for why smokers smoke more when stressed. According to Schachter (*42*), the debilitating effects of no or low nicotine are the result of withdrawal, and the effect of stress is to put the smoker into withdrawal by depleting the available supply of nicotine. This hypothesis was strengthened and new leads were generated by biochemical studies showing that, while some nicotine is catabolized (mainly in the liver, at a constant rate determined in part by the duration of the habit), a fraction of the nicotine escapes detoxification and is eliminated directly in the urine. Furthermore, the rate of urinary excretion is rapid, increases linearly with dosage, and increases as the pH of the urine becomes more acid. The hypothesis was confirmed by direct manipulation of urinary acidity through the administration of mild acidifying agents like ascorbic acid or glutamic acid hydrochloride or alkalizers like sodium bicarbonate (*43*). In addition, stressful events associated with heavier smoking increased urinary acidity and nicotine excretion in the expected direction (*42*). To test whether stress or urinary pH or both were the independent variable, Schachter et al. (*43*) independently manipulated stress and pH and reported that smoking seemed to be under the control of urinary acidity rather than stress as such.

Schachter's model posits that nicotine is the primary reinforcer because of its role in reducing tension and distress associated with nicotine deprivation. If this is true, secondary reinforcers should be relatively unimportant. For example, smokers should not smoke nicotine-free cigarettes, and supplying alternative sources of nicotine should eliminate the desire to smoke. According to Jarvik (*19*), much of the evidence for the role of nicotine as the primary reinforcer in cigarette smoke is circumstantial. Smokers evidently prefer cigarettes with, rather than without, nicotine; but they will smoke nicotine-free cigarettes for a while if no others are available. The fact that smoking such cigarettes is not sustained despite the usual cues for smoking suggests that the other variables are secondary reinforcers that extinguish when nicotine—the primary reinforcer—is not present. Attempts to investigate the role of nicotine as the sufficient condition for smoking, however, have produced conflicting results. Preloading nicotine, by having subjects smoke or chew gum containing nicotine before testing, did reduce subsequent puffing (*20, 21, 25*). And administration of the drug mecamylamine, which functioned as a nicotine "antagonist," increased the smoking rate (*52*). But Kumar, et al. (*21*) were unable to demonstrate a dose-response effect on subsequent smoking when nicotine preloading was administered intravenously. The fact that lettuce cigarettes reinforced with nicotine were as unacceptable as non-nicotine cigarettes also seems to undermine the nicotine-only hypothesis (*19*). Jarvik (*19*) concluded that nicotine may be a *necessary but not sufficient* condition for smoking behavior to occur and to be sustained and that more research is clearly needed to settle the issue of whether nicotine functions as the primary reinforcer or as a "reinforcing co-factor."

The nicotine addiction model suggests that the smoker regulates nicotine levels under widely varying conditions. It implies a mechanism which senses nicotine and provides the impetus for directed behavior—possibly a central "nicostat" or the integration of the various peripheral drug effects of nicotine. While the model is plausible and straightforward, critical tests have yet to be performed. Particularly, direct measurements of changes in nicotine titer and of the withdrawal state have not been attempted. Finally, among variables not adequately explained by the model are the role of environmental stimuli in the control of the habit, the nature of individual differences in smoking behavior (for example, light versus heavy smokers and occasional versus chronic smokers), and the mechanism(s) by which relapse occurs following withdrawal (*35*).

A Context for Behavioral Research on Smoking

Clearly, neither social learning theory nor the nicotine addiction model alone can provide a complete understanding of smoking at present. A recent model, the opponent process theory (*47, 48, 49, 53*) does attempt to link psychological and physiological factors involved in the maintenance of smoking in a more comprehensive fashion. The principal features of the opponent process model as it applies to smoking are as follows: (*1*) the reaction to cigarette smoke is biphasic, with a brief pleasurable component (*a* process) followed by a more sustained dysphoric component (*b* process); (*2*) the hedonic tone—pleasurable A state or dysphoric B state—is determined by the algebraic sum of the two opponent processes at a given point in time: and (*3*) stimuli associated with a given state can elicit this state as a conditioned response after repeated pairings.

The opponent process model assumes that cigarettes contain substances which provide pleasure (initiate the *a* process) during early use. While there may be some unpleasant effects on the first few occasions, these should be offset by the drug effect or by other reinforcers such as peer pressure; if not, the act of smoking will not continue. As cigarette smoking becomes established, the opponent process grows in strength: the pleasurable A state weakens and the withdrawal B state intensifies correspondingly.

Because the *b* process is the opponent of the *a* process, the best way of attenuating the B state is to ingest the substance that produces the A state. As an operant behavior, smoking is both positively reinforced by a pleasurable consequence and negatively reinforced by terminating aversive withdrawal, thus setting up an addictive cycle. As the *b* process is further strengthened, still larger amounts of tobacco have to be smoked to produce a pleasurable A state, resulting in tolerance.

Stimuli associated with smoking (CS_A's), such as a pack of cigarettes or the sight of matches, should elicit a brief conditioned (pleasurable) A state at stimulus onset and a conditioned withdrawal (unpleasant) B state at stimulus offset. Furthermore, stimuli associated with the B state (CS_B's)—such as an empty cigarette pack, empty pockets, no stores, or "no smoking" signs—should elicit conditioned craving or withdrawal. The concept of conditioned A and B state elicitors leads to the important implication that, as the smoking habit becomes well established and the *b* process becomes stronger, CS_A's elicit a brief conditioned state which is pleasant but then is followed by a more extended conditioned craving which intensifies the pre-existing withdrawal B state. Similarly, CS_B's directly elicit conditioned craving, which also adds to the discomfort of the withdrawal state. An additional implication (derived from Pavlovian conditioning theory) is that as CS_B's become stronger, they may become more anticipatory, leading to shorter redosage and restimulation intervals until an asymptote is reached. If the smoker quits, the CS_B's and the *b* process should weaken eventually through disuse, but the CS_A's and the *a* process should intensify correspondingly. Thus, if a cigarette is smoked after a period of abstinence, the pleasurable component has increased to its original level and the resumption of the addictive cycle is facilitated. The smoker is clearly locked into the pattern of smoking and, in that sense, once established, the habit seems to be overdetermined.

The opponent process model has not been tested in formal research on cigarette smoking, though recent experiments in the area of opiate addiction do provide general support (*31*, *44*, *56*). The demonstration of conditionability, in particular, has important implications for the understanding of smoking recidivism. Wikler (*55*) has observed that environmental stimuli associated with withdrawal may precipitate conditioned craving (or withdrawal) even after an extended abstinence period has ended physical dependence in heroin addicts. The opponent process model predicts a biphasic response by smokers (A state followed by B state) to the presentation and removal of stimuli associated with cigarettes during acquisition. Later on in the addiction process, when tolerance is large, the dominant conditioned effects should be those of craving or withdrawal (B state predominates). The implication for treatment is that unless conditioned craving is extinguished or modified as a part of therapy, the probability of relapse will remain high.

There are a number of different issues that need to be resolved among the current behavioral formulations of smoking before an adequate understanding is achieved. For example, the nicotine addiction model suggests that the day-to-day regulation of smoking is more under the control of pharmacological variables than of environmental stimuli, though their relative contribution remains to be determined. Moreover, the issue of whether smoking reduces anxiety is not settled. For example, Hutchinson and Emley (*18*) have suggested that nicotine can be classified as a tranquilizer since it decreases aggression as well as the conditioned emotional response (CER). They have speculated that difficulty in training animals to smoke under ordinary conditions may have been because a background of aversive stimulation is needed to provide motivation to use smoking to relieve anxiety. Also, as has been mentioned, the pharmacological primacy of nicotine implied by the nicotine addiction model has yet to be established unequivocally.

The opponent process model encounters similar problems. For example, Wikler (*55*) has argued that certain responses associated with chronic drug use, such as tolerance or conditioned withdrawal, are *counteradaptations*, serving to protect the organism by acting in a direction opposite to the normal drug effect. The opponent process model is stated in sufficiently general terms to incorporate these observations if certain (untested) assumptions are made: Wikler's observations emphasize the dominant drug-negative B state; in opponent process theory, the initial drug-positive *a* process (and thus the pleasurable A state) is still operative but may be so brief and attenuated that it goes undetected. Only closer examination of the time course for the response to drugs at different states of acquisition will settle this issue. An additional complication has been raised by Siegel (*45*), who has shown that the stimuli which constitute the ritual of (repeated) drug injection can elicit conditioned reactions which increase tolerance to the drug; extinction of these conditioned reactions, using a series of saline injections, results in decreased tolerance. Siegel proposes that tolerance is the result of compensatory *associative* processes and is not simply a pharmacological, nonassociative phenomenon. While opponent process theory can be modified to accommodate these findings, by defining them as the manifestations of stimuli which serve as conditioned B state elicitors, the relative contribution of associative and nonassociative factors cannot be specified at present. Furthermore, if tolerance is basically an associative process, the problem of explaining why certain substances, such as nicotine, produce tolerance while others do not will also have to be dealt with (*35*).

The remainder of the present discussion will re-examine some of the phenomena of acquisition, perpetuation, and termination of smoking from the point of view of the three models. Special attention will be given to implications for further research.

The Establishment of Smoking

The establishment of smoking can be seen as the result of initial experimentation with cigarettes repeated sufficiently often for acquisition of a habit and/or for addictive processes to take hold. Among the major variables contributing to initiation are social pressure and imitation of peers or family members who smoke (*1, 11*). The following variables influence the decision to smoke: peer pressure, best friends who are smokers, parents who smoke, adolescent rebellion, imitation of adult behavior, and misconceptions concerning the risks of smoking. A recommendation to conduct longitudinal comprehensive studies on the acquisition of smoking in the natural environment, and to determine the conditions under which smoking does or does not begin, would seem especially appropriate.

Once the smoking habit is acquired, the stage is set for addictive processes to contribute to the maintenance of the habit and to its overdetermination under the influence of the variables alluded to in the several smoking models. Additional physiological variables and explanatory variables from personality theory and typology studies (both types described elsewhere in the present report) are clearly relevant. These two sets of variables suggest a number of possible mechanisms by which acquisition might take place, although, as Leventhal and Cleary (*22*) point out, they are not necessarily the same mechanisms which contribute to onset. The need for careful, directed research in this area is evident to achieve a better understanding of onset and acquisition which may lead to more effective methods for prevention and treatment.

A promising approach to the investigation of physiological and behavioral, as well as psychosocial, factors in acquisition comes from animal research. Some studies have shown that nicotine facilitates conditioned-avoidance behavior as well as positively reinforced behavior in rats (*51*) and that it reduces social or pain-induced aggression in both animals and humans (*18*). Analogues of addiction might also be explored in the laboratory. While the laboratory approach might seem artificial to some, increasing experimental control by restricting extraneous variables has been useful in other difficult areas, such as alcoholism (e.g., Nathan and O'Brien (*29*)) and heroin addiction (e.g., O'Brien, et al. (*32*)). If such explorations are successful, subsequent research could be conducted under increasingly complex and more "natural" conditions. Finally, studies of different methods for deterring smoking in children (e.g., Evans (*7*) and Piper (*34*)) should increase understanding of the conditions under which smoking begins and allow us to identify those environmental patterns which facilitate the movement from "experimental" smoking to addiction.

The Maintenance of Smoking

Once smoking is established as a habit, a number of factors contribute to its persistence and resistance to change. Each of the formulations described above devotes considerable attention to the phenomenon of maintenance, and a large body of research has been carried out from various points of view. In a sense, maintenance can be seen as a stage of smoking characterized by steady-state behavior. Pattern consistency is provided by environmental influences through stimulus control as well as by underlying physiological processes regulating consumption within characteristic limits. As an acquired motivation, smoking constitutes a behavioral pattern with powerful reinforcing value, overdetermined to a remarkable degree by its generating mechanisms. A better understanding of these processes is needed.

With a few exceptions, the determination of environmental influences on smoking has received relatively little direct attention experimentally, despite the fact that treatment techniques based on social learning theory have been used extensively. Among the better examples of a functional analysis of behavior is a study by Griffiths, et al. (*12*). Following detoxification, alcoholics in a residential laboratory were allowed to consume ethanol at certain times, and the amount of tobacco smoked was measured under various conditions. Cigarette smoking was shown to increase from 26 to 117 percent when the solutions consumed contained ethanol. The effect was robust, was observed in each of the five subjects, and was replicated 15 times employing a within-subject design. Control procedures indicated that the effect did not depend on: (1) the pattern of ethanol ingestion, (2) adjunctive maintenance through social interactions, (3) the pattern of days in which the ethanol or ethanol-free vehicle was scheduled, (4) alterations in the portion of cigarette smoked or the number of puffs taken, or (5) knowledge that a given drink did or did not contain ethanol. The study constitutes a good demonstration of the potential of the experimental analysis of smoking behavior, and the method should be extended to other problems of interest.

Smoking as an avoidance/escape response to withdrawal implies an internal regulatory mechanism by which the levels of nicotine (or other substances) are maintained within limits characteristic for each smoker. To get at these processes in research, measures should be taken of smoking behavior (specifying variables such as puff frequency and duration, depth of inhalation, amount of nicotine drawn from a standard cigarette), of major physiological variables (for example, cardiovascular changes, relevant biochemical activity including cholin-

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ergic, catecholamine, and nicotine changes), and of cognitive variables (for example, hedonic states and the subjective desire to smoke at different points in time). As in investigations on the establishment of smoking, a laboratory approach may provide a good initial strategy, if supported by adequately controlled studies in the natural environment.

As a preliminary step, the variables involved in nicotine regulation should be explored directly in habitual smokers by studying the relationships between the act of smoking, subjective desire, and plasma nicotine levels. Also, nicotine excretion rates could be shifted using techniques identified by Schachter, such as drugs or psychological stress, to provide further modulation of physiological, behavioral, and subjective responses, thus replicating and extending previous work in this area. The demonstration of the contribution of nicotine by direct measurement might stimulate further explorations of the relationship between smoking behavior and other important biochemical variables such as catecholamines.

The Cessation of Smoking

Both initiation and cessation can be conceptualized as the result of decisions (evidenced by stated intention or other overt behavior) to start or to stop smoking. Thus, cognitive variables may play a major explanatory role, and the subjective utility of the change under consideration may provide important clues for predicting its outcome or success (*33*). (The cognitive aspects of initiation and quitting are extensively reviewed in a separate context elsewhere in this report.) Once the decision to start or stop smoking has been made, however, behavioral variables and the models described above come into play.

When habitual smokers stop smoking, they may experience a wide variety of unpleasant side effects, including craving for tobacco, irritability, restlessness, dullness, sleep disturbances, gastrointestinal disturbances, anxiety, and impairment of concentration, judgment, and psychomotor performance (*19*). The onset of symptoms may occur within hours or days after quitting and may persist from a few days to several months. Additional objective signs include a decrease in heart rate and blood pressure, increased rapid eye movement (REM) sleep, and slower rhythms in the EEG (*35*). Spontaneous jaw clenching (increased masseter potentials) lasting several weeks has been correlated with verbal reports of irritability (*18*).

After the ex-smoker successfully overcomes withdrawal symptoms, further problems may persist. In terms of the opponent process model, one can construct the following account: Subjectively, the pleasure of smoking in the addicted smoker is masked by the discomfort of craving from not smoking. After abstaining for a few weeks, however, craving decreases. If smoking is resumed, the first few cigarettes seem very strong and are highly pleasurable. Thus, the stage for re-addiction is set. Moreover, various internal and external stimuli may serve as conditioned elicitors of craving or withdrawal. Particularly troublesome may be events too infrequent to extinguish quickly (e.g., attending a reunion where former classmates smoke) or emotional situations which resemble withdrawal (e.g., anticipation of an unpleasant or challenging social event).

A major contribution of the behavioral approach has been the development of new techniques in smoking cessation—procedures which seem to be more effective than those that preceded them. In most nonbehavioral clinics, fewer than half the smokers quit (e.g., Guilford (*13*)), and of those who quit only 25 to 30 percent are still abstinent 9 to 18 months later (*17*); the estimated long-term abstinence rate in nonbehavioral treatment is about 13 percent (*27*). The three main lines of behavioral treatment have involved punishment and aversive conditioning, stimulus control and contingency management, and controlled smoking procedures. While a thorough review of the modification of smoking is provided elsewhere in this report, the contribution of social learning to therapy is of sufficient importance to warrant a brief review here.

Aversive conditioning techniques are the oldest and most widely utilized behavioral procedures for smoking cessation. Among the aversive stimuli used have been electric shock (e.g., Best and Steffy (*3*)), covert or imagined aversive events, and cigarette smoke (e.g., Resnick (*39*)). The typical procedure has involved contingent punishment for overt smoking behavior in the laboratory or in the natural environment (e.g., Powell and Azrin (*38*)). Some investigators have attempted to punish motoric and cognitive components as well (e.g., Steffy, et al. (*50*)). With the exception of aversive smoking procedures, aversive conditioning techniques have not produced outstanding results (Bernstein and Glasgow (*2*)).

Aversive smoking combines the principles of extinction, negative practice, and aversive conditioning, using stimuli from the cigarettes themselves as the aversive component. The procedure assumes that the positive reinforcing aspects of a stimulus are reduced and become aversive if that stimulus is presented at an artificially elevated frequency or intensity. A further assumption is that aversion based on stimuli intrinsic to the maladaptive behavior is more salient and generalizable than that from artificial sources such as shock (Bernstein and Glasgow (*2*)). The most successful use of aversive smoking can be found in the recent work of Lichtenstein, et al. (*24*), using a technique called rapid smoking. The procedure calls for smoking cigarettes at a rapid rate (inhaling smoke about 6 seconds after each exhalation) until no more can be tolerated. Sessions are repeated on a daily basis until the smoker no longer reports a desire to smoke; booster sessions are provided if the desire returns. In a recent review of several studies using the procedure, the abstinence rate was 54 percent in short-term follow-up and 36 percent in long-term follow-up (2 to 6 years after treatment). Though the method was a clear improvement over previous approaches, there are a number of problems which may make it less than the optimal procedure for the elimination of smoking. In particular, individuals with cardiopulmonary diseases—those who most need help—are the least likely to tolerate intense exposure to tobacco smoke without ill effect (*35*). Moreover, rapid smoking may be dangerous even to seemingly healthy people (*28*).

Another social learning approach to the modification of smoking behavior is represented by stimulus control tactics. The basic assumption is that smoking is associated with or controlled by environmental cues and that these cues (discriminative or conditional stimuli) contribute to the persistence of the habit (*2*). Treatment involves gradual elimination of smoking through programmed restriction of the range of stimuli that lead to smoking. Typically, self-monitoring is used to increase awareness of smoking along with designated daily quotas to provide targets for reduction (*36*). In general, stimulus control procedures have not been very effective in isolation (e.g., Levinson, et al. (*23*)). When used in combination with contingency contracting, in which deposited money is reimbursed for reaching specified goals (e.g., Elliott and Tighe (*6*)), and with other techniques, however, considerably better results are achieved (Bernstein and Glasgow (*2*)).

Recent research on multicomponent treatment procedures (employing techniques such as stimulus analysis, interference with situational control or environmental stimuli, social and monetary reinforcement of incompatible behavior, group support, and follow-up sessions, presented in an integrated sequence) has produced results as favorable as that reported for rapid smoking, with 61 percent of the first 100 participants quitting smoking after eight sessions of treatment and 32 percent not smoking a year after the onset of treatment (*36*). These data account for all smokers who entered treatment (including the 15 percent of the sample who could not be reached and were classified as smoking) and were based on self-reported smoking status corroborated by urinary nicotine analysis. The recidivism rate of 49 percent also compares favorably with the 70 to 75 percent recidivism reported for nonbehavioral clinics by Hunt and Bespalec (*17*). These positive findings are qualified somewhat by the observation that not all multicomponent treatment combinations are successful (e.g., Danaher (*5*)) and by a controlled multivariate study by Flaxman (*8*) indicating that the variables responsible for a successful outcome are poorly understood.

Smoking practices have changed considerably in recent years as smokers have attempted to reduce health risks on their own (Hammond, et al. (*14*)) by switching to filtered and low tar/nicotine cigarettes (Russell (*41*)). These natural trends provide a context for recent research by Frederiksen and associates (*9*, *10*), demonstrating that behavioral technology can be used to control not only the rate and strength of cigarettes consumed but also to modify the topography of the habit. Additional impetus for the research comes from the fact that many smokers report difficulty reducing their smoking rate below 10 to 12 cigarettes per day (Levinson, et al. (*23*)). While it has been suggested that the reason for this is that the positive reinforcing value of each cigarette increases when fewer are smoked (Mausner (*26*)), according to opponent process theory there should be a corresponding lessening of the negative reinforcing effect resulting from withdrawal from nicotine over time. Clearly more research is needed to settle this issue. The technology developed by Fredericksen is still in the clinical development stage, and the long-term stability of the changes has yet to be determined. However, because some smokers are motivated to reduce their health risk even though they are unable to quit, controlled smoking technology may provide a useful alternative to the more traditional abstinence-oriented treatment and deserves further exploration.

While recent behavioral treatment seems more effective than previous approaches, 50 percent recidivism and 33 percent long-term abstinence leave considerable room for improvement. What is needed at present is outcome research directed at demonstrating the relative effectiveness of complete treatment packages in long-term randomized clinical trials. Subsequently, when a given procedure is shown to be superior in independent replications, components can be partitioned out and tested in order to produce clinical procedures that are both effective and efficient. Research designs should take into account the fact that recent improvements in outcome statistics for smoking-cessation clinics may reflect changing social attitudes toward smoking and higher levels of motivation rather than better treatment as such (*22*).

In an important sense, current treatment efforts—especially behavioral treatment—have been devoted primarily toward the modification of the overt act of smoking (an operant behavior). Less formal attention has been given to the cognitive and physiological respondents that constitute precursors of smoking (e.g., craving and withdrawal) and that are under the control of both environmental (exteroceptive) and emotional (interoceptive) stimuli. Moreover, the increased success of multicomponent programs may well be the result of more effective handling of these variables, using integrated sequences, than has been possible with unicomponent approaches. The fact that various previously neutral stimuli have been shown to elicit conditioned craving or withdrawal after being paired or associated with these states in various addictions has important implications for smoking treatment.

Treatment can be seen as extinguishing the act of smoking but not necessarily the concomitant conditioned cognitive or physiological respondents. As a result, the ex-smoker may continue to be exposed to various stimuli which have been associated with smoking, and the probability of relapse will remain great (for example, in the "negative affect" smoker (*36*)). Demonstrations that continued autonomic or cognitive reactivity persist after standard smoking-cessation therapy might lead to an entirely new approach to the old problem of relapse. Studies comparing a standard smoking-cessation treatment with "deconditioning" therapy, in which autonomic responses are extinguished in a simulated environment or modified directly using biofeedback, might lead to a demonstrably lower rate of recidivism for those smokers exposed to augmented therapy. The above suggests that basic research which leads to a better understanding of the mechanisms underlying smoking may result in the eventual development of a truly rational and more effective therapy for smoking.

Conclusions

The present chapter makes no claim to be exhaustive. Rather it has surveyed selectively what is known and not known concerning behavior in the establishment, maintenance, and cessation of smoking. The object has been to develop a context for directing research, for improving treatment, and for guiding social policy. In closing, a few specific recommendations seem appropriate.

While it is difficult to pinpoint accurately which of many research possibilities will be most fruitful on an *a priori* basis, certain themes seem particularly important for current behavioral research. They are the phenomenon of withdrawal, the reinforcing effects of nicotine, the role of nicotine antagonists or blockers, and the behavioral pharmacology of cigarette smoking.

1. Withdrawal symptoms of varying severity following cessation are among the principal reasons cited for relapse to smoking. Little scientific information is available on the sequelae to abstinence, however, and at present it is difficult to assess accurately their contribution to recidivism.

2. As discussed at some length, the problem of analyzing the reinforcing effects of nicotine is of great importance in understanding smoking. The role of nicotine as a positive and negative reinforcer should be examined in animals using various routes of administration as well as explored systematically in humans in laboratory and natural settings.

3. A related theme is derived from recent research suggesting that specific CNS receptor sites for nicotine can be blocked in a fashion analogous to the opiate antagonists. This phenomenon has implications

for understanding the effect of nicotine on the body as well as in helping smokers who have stopped to maintain abstinence.

4. The behavioral pharmacology of smoking deserves further emphasis. A more precise definition of smoking behaviors, involving psychometric analyses by puff volume, inter-puff interval, total amount smoked, and rate of smoking may have important implications for the understanding of stimulus control as well as of the relationship between blood nicotine levels and cigarette self-administration. Similarly, the development of objective criteria for validating dependent measures (such as self-reported smoking behavior using various biological assays) seems worthwhile.

In the treatment area, further improvement is clearly needed. Multicomponent procedures have provided sequences for handling different aspects of the smoking-cessation process; and components dealing specifically with problems in measuring baseline smoking, facilitating reduction, inducing abstinence, and managing side effects have been developed. Among the major current deficits for all approaches and programs, however, is maintenance of nonsmoking. Several suggestions have been made from a behavioral point of view. These include: (1) dealing promptly and effectively with the potential side effects of quitting (such as obesity and tension); (2) developing alternative activities to replace smoking (such as regular physical exercise or formal relaxation techniques); (3) providing a cognitive focus on mastery, self-help, and individual responsibility; and (4) adding "booster" sessions and continued interpersonal support in extended follow-up. Much more remains to be done—especially on the utilization of techniques derived from basic research, such as the extinction of conditioned craving described above.

Behavioral research may also make contributions to social policy. For example, the suggestion that nicotine plays a major or dominant role in the self-regulation of smoking raises the issue of the appropriateness of trying to persuade people to smoke low-tar, low-nicotine cigarettes. As Schachter (*42*) puts it, low-tar, high-nicotine cigarettes might be safer because fewer cigarettes would be smoked, thereby minimizing exposure to the products of incomplete combustion known to enhance the atherosclerotic process and to increase the risk of myocardial infarction (*19*). This problem could be investigated further, using a careful description of the number of cigarettes smoked and the number of puffs per cigarette (backed up with quantitative determinations of nicotine, carbon monoxide, tars, and other smoke products), to provide more exact information than is currently available from surveys of smoking in the natural environment. Finally, a greater understanding of the stimulus control of smoking and its limits may be very valuable. From a behavioral perspective, the current growing emphasis on the social unattractiveness of smoking (for example, the nonsmoker's rights movement) is helpful, because it provides a method which administers more immediate social reinforcement for quitting and staying off cigarettes than has been possible when the focus was strictly on the health consequences of the habit. It should be noted that the effects of these social processes on the decision to quit smoking are still relatively underexplored.

Much work remains to be done in the behavioral research area. Sufficient progress has been made, however, to indicate that the development of a rational therapy for smoking based on a scientific understanding of smoking behavior and its underlying mechanisms constitutes a worthy objective.

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Smoking in Children and Adolescents: Psychosocial Determinants and Prevention Strategies

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Introduction

In spite of a decrease in adult smoking since the dissemination of the 1964 U.S. Surgeon General's Report on Smoking and Health, there is discouraging evidence that smoking among teenage boys is remaining virtually constant and among teenage girls it is actually increasing. It is apparent that more knowledge is needed concerning the way in which the psychosocial factors that may contribute to the initiation of smoking can be applied to the development of effective strategies to deter the onset of smoking.

It is possible that prevention programs directed at children and adolescents have generally placed too much confidence in merely communicating knowledge about the dangers of smoking. Developers of these programs may assume that such fear arousal will in itself be sufficient to thwart smoking. In fact, as will be amplified later in this chapter, by the time children reach junior high school, almost all of them believe smoking is dangerous. It appears that communications concerning the dangers of smoking whether delivered from schools, churches, voluntary agencies, mass media, the family, peers, governmental agencies, industrial organizations, consumer organizations, or labor unions (individually or collectively) have, indeed, been effective in persuading children and adolescents that smoking is dangerous. However, it is also evident that fear of the consequences of smoking may in itself not be sufficient to discourage a substantial number of children from beginning to smoke when they approach adolescence.

Some investigators in this field have contended that at an earlier level of the child's development, perhaps between the ages of 4 to 9 or 10, the child takes quite literally the dangers of smoking. In fact, it is often observed at this level of development that children may be especially worried if they observe a parent or older sibling smoking. They will admonish them to stop smoking because it "can cause cancer or a heart attack." Yet as they approach adolescence, many of these same children will begin smoking.

Responses from the teenagers themselves suggest that peer pressure to smoke may be one of the major influences. There is also some evidence that the smoking parent becomes a model for the child. If both parents smoke there is a greater likelihood that the child will begin smoking than if only one parent smokes or if neither parent smokes. But even if one parent smokes, this may influence the child to smoke more than if neither parent smokes. Interestingly, if an older sibling and both parents smoke the child is about four times more likely to smoke than if there were no smokers in the family.

The influence of the mass media in the initiation of smoking is somewhat more difficult to establish. Smokers are depicted in films and television, as well as in cigarette advertising which tends to portray them in interesting and exciting environments, suggesting that attractive, desirable people tend to smoke. This would logically be expected to influence children and teenagers much as the media and advertising affect the behavior of adults. Yet, the relationship between exposure to the mass media and the initiation of smoking is difficult to isolate from the other concurrent influences to which the child is exposed. In fact, a variety of psychosocial influences may interact to influence some children to begin smoking.

Some investigators examining the issue of why fear arousal may often have such a limited effect on health behavior suggest that much of the information communicated to children concerning smoking and its dangers may be too general and not sufficiently personalized. Also, the suggested harmful effects of smoking in many smoking control messages violate the concept of "time perspective." As children grow older they recognize that people around them who smoke do not die instantly and that heart attacks or cancer are not a certainty. They may need to be exposed to evidence that smoking has immediate physiological effects on the body. Younger adolescents particularly live in the present and are not preoccupied with the future. Emphasizing what might happen to them when they are much older may not be an effective way to persuade many of them to resist the pressures to begin smoking.

Becoming a smoker may have the immediate value to some teenagers of being accepted by their peers, feeling more mature because smoking is an adult behavior forbidden to the child, providing a level of physiological stimulation and pleasure, and might even serve the function of an act of defiance to authority figures. The prevention programs reviewed rarely incorporate such concepts. Rather, they focus primarily on information relating to the long-term dangers of smoking.

Furthermore, too few of the prevention programs are evaluated with sufficient rigor. As a result, in the same sense that there is insufficient basic behavioral research to link clearly many psychosocial factors to the initiation of smoking in children and adolescents, it is difficult to determine if many prevention programs significantly deter the onset of addictive smoking. Even if a program results in increased knowledge concerning the long-term dangers of smoking, in the absence of valid evidence of a direct impact on the incidence of smoking itself, it is possible that many widely disseminated prevention programs are, in the long-run, of only questionable value in actually deterring smoking. All of this suggests many avenues for future research and prevention programs.

To elaborate on the various points discussed above, the sections which follow deal with current smoking patterns and beliefs, relevant conceptual models in developmental and social psychology, typical psychosocial influences in the smoking decision, critical evaluations of some current prevention programs, and finally, some recommendations for future research and prevention programs.

Current Smoking Patterns and Beliefs

While cigarette smoking in the United States for adults over age 21 has declined, there has been a growth in the amount of smoking among the pre-adult population, primarily due to a dramatic increase in smoking among teenage girls (*61*). But care needs to be exercised when interpreting the findings of the studies reported since definitions of such terms as "regular smoker," "occasional smoker," "experimental smoker," and "nonsmoker," vary from one study to the next. For example, four national surveys conducted at 2-year intervals from 1968 through 1974 by the National Clearinghouse for Smoking and Health (*61*, *86*) define a current regular smoker as one who smokes one or more cigarettes per week. On the other hand, an antismoking education study conducted at the University of Illinois (*18*) defines a current regular smoker as one who smokes cigarettes just about every day. Also contributing to the ambiguity of results is the way in which the categorization of frequency of smoking is dealt with in the analysis of results. For example, in the four national surveys previously cited, experimental smokers (those who have smoked at least a few puffs but less than one hundred cigarettes) were combined with nonsmokers in the analysis of the data. Experimental smokers are extremely important and should not be neglected in data analysis since experimental smoking is obviously the initial step toward confirmed smoking (*42*).

In the four surveys (*61*) conducted by the National Clearinghouse, approximately 16 percent of the teenage population, aged 12 to 18, were current regular smokers in 1974. The rate of regular smoking for the same age group in 1968 was approximately 12 percent. In the first survey, only about half as many girls as boys regularly smoked, but by 1974 this difference had virtually disappeared. In fact, regular smoking had slightly decreased for boys from 1970 to 1974, but this decrease was easily offset by the dramatic rise in smoking by girls.

Relevant to the problem of teenage smoking is the age of initiation of smoking. A significantly larger percentage of regular smokers aged 12 to 14 were reported among teenagers in 1974 (approximately 12 percent) than in 1968 (approximately 6 percent). This increase in regular smoking at younger ages suggests that the average age of the initiation of smoking is decreasing.

Further evidence concerning the age of initiation of smoking is available from retrospective data reflecting self-estimates of onset of smoking in the Current Population Surveys of 1955 and 1966 (*1*). No analysis of age trends in smoking initiation among males was reported since the number of male respondents was low, particularly in the 1966 survey. However, the responses from the female respondents, regardless of their current age, suggest a shift in the initiation of smoking to a younger age. For example, over twice as many females, aged 18 to

24, classified themselves as regular smokers by age 15 in 1966 than did the respondents of the same age group in 1955.

In the national surveys between 1968 and 1974 (*61*) the relationship between various factors related to socioeconomic status and smoking were examined. For example, teenagers who are employed outside the home are twice as likely to smoke as teenagers who are not employed. Also, educational and vocational aspirations are related to smoking. Students who plan to go to college are the least likely to smoke. A study conducted by Borland and Rudolph (*9*) determined that socioeconomic status bears some relationship to smoking in high school students (children in lower socioeconomic levels are more likely to smoke), but socioeconomic status correlates less with smoking than parental smoking or poor scholastic performance (although all three variables are themselves correlated).

The literature fails to address adequately the initiation of pre-adult smoking. Rather, the emphasis is on "regular" smokers. Nevertheless, inferences from such data may be helpful in suggesting factors that are related to the initiation of smoking.

As would be expected, beliefs of teenagers about smoking are related to whether or not they smoke. Of course, smokers generally hold more favorable attitudes toward smoking than do nonsmokers (*65, 75*). Nevertheless, data (*59*) suggest that even teenage smokers seldom consider the decision to smoke a wise decision. For example, 77 percent of smokers believe that it is better not to start smoking than to have to quit. Over half of the teenage smokers believe that cigarette smoking becomes harmful after just 1 year of smoking. Eighty-four percent say it is habit forming, while 68 percent agree that it is a bad habit. Of all teenagers, 78 percent believe that cigarette smoking can cause lung cancer and heart disease. Eighty-seven percent of all teenagers and 77 percent of teenage smokers believe that smoking can harm their health. The vast majority of teenagers consider smoking as habit forming, but almost two-thirds do not feel that becoming addicted to smoking is an imminent threat to their health. Experimental smoking is considered safe.

Fishbein (*34*) cites evidence from a study conducted for the American Cancer Society in 1975 which suggests that teenage smoking is perceived by teenagers as more prevalent than it actually is. Eighty-three percent of the teenagers in this survey tend to think of other teenagers as being smokers rather than nonsmokers.

Finally, it should be pointed out that knowledge or beliefs about the dangers of smoking are often confused with attitudes toward smoking (*10*). Attitudes may be much more complex than simple beliefs about the harmful effects of smoking. Various factors influencing the complexity of attitudes toward smoking are discussed in the most recent report of the four national surveys mentioned earlier (*61*). These factors include the adverse effects of smoking on the individual's health and on the environment (pollution), the psychological and sociological benefits of smoking (e.g., "makes you feel good"), rationalizations that allow smoking, perceptions of reasons for smoking and for smoking initiation, the negative stereotypes concerning smokers, attitudes toward authority, and control over one's destiny.

In essence, when considering both current smoking patterns and beliefs among children and adolescents, the factors related to smoking can be categorized in terms of perceived psychosocial benefits versus actual threats to health. Considering this dichotomy, the suggestion of the U.S. Public Health Service (*61*) should not be ignored:

> It is futile to continue to tell teenagers that smoking is harmful and that they shouldn't do it. They know that it is harmful. Most do not *want* to do it. The most effective thing that we can do is to help them to understand the benefits of smoking as compared with the costs and dangers so that they will have the facts that they need in order to make a thoughtful decision as to whether to smoke or not to smoke (p. 27).

Relevant Conceptual Models in Developmental and Social Psychology

Understanding the factors involved in the initiation of smoking among children and adolescents is a complex endeavor demanding the utilization of diverse conceptualizations. This section will consider four representative conceptual models in developmental and social psychology that would appear to be potentially useful in generating hypotheses to account for the initiation of smoking among the young and in providing conceptual bases for prevention programs. These conceptualizations are Piaget's Cognitive Development Theory, Erikson's Theory of Psychosocial Development, Bandura's Social Learning Theory and McGuire's Persuasive Communication Model.

The Cognitive Developmental Theory of Piaget (*26, 69*), one of the most influential cognitive theories, is concerned with the nature and origin of knowledge. Piaget's view of the development of knowledge would appear to offer some applications to understanding the informational and decisional aspects of the initiation of smoking in the developing child.

Piaget views knowledge as developing out of the individual's adaptive interaction with the environment through the processes of assimilation (incorporation of concepts into existing cognitive structures) and accommodation (modification of cognitive structures). There are four major stages of intellectual development: (1) sensory-motor period (birth to 2 years), involving simple perceptual and motor adjustments to immediate environmental phenomena; (2) preoperational period (2 to 7 years), involving a preconceptual phase (the emergence of linguistic skills and symbol construction abilities) and an intuitive phase (the emergence of more complex thoughts, images, and classification abilities based on perceptual similarity instead of logical considerations); (3) concrete operational period (7 to 11 years), involving reversible intellectual operational ability (utilizing a mental representation of a series of actions), conservational ability (realizing that quantity remains invariant despite perceptual transformations), a clearly defined concept of class inclusion, and the ability to take the viewpoint of another; and (4) formal operational period (11 to 15 years) involving the realization that reality is but one of a set of all possibilities. Thinking in this last stage is characterized by hypothetical-deductive reasoning, combinational analysis (consideration of multiple factors), propositional and rule-governed logic, and a futuristic perspective.

Piaget's ideas, especially those dealing with developing knowledge about the physical environment, have been extensively explored, although the investigation and application of his concepts involving adaptation to the social environment have only rarely been studied. The initiation of smoking, apparently an age-related behavior, appears most often to occur within the context of social interactions. Additionally, smoking involves an important decisional component requiring the utilization of cognitive or knowledge structures.

By the time they reach the seventh grade, the vast majority of children believe smoking is dangerous to one's health (*31*). Yet despite this knowledge, many adolescents, aged 12 to 14, experiment with smoking, and roughly 4 to 5 percent will smoke regularly (weekly) (*61*). This situation suggests that "social adaptation" may override "intellectual adaptation" or knowledge. Knowledge of the dangers of smoking often motivates a preadolescent to become a crusader against smoking, while the social pressures occurring during early adolescence may outweigh the effects of this concrete knowledge. So, the individual who had been at an earlier age an antismoking crusader may become a regular smoker or at least an experimental smoker as a teenager. This conflict between knowledge of the dangers of smoking and smoking suggests the possibility of observing the development of smoking within the Piagetian framework.

One contemporary psychoanalytic developmental model of consequence is Erikson's Theory of Psychosocial Development (*24*, *25*) involving eight psychosocial crises. These crises are: (1) trust vs. mistrust (0 to 1 year), (2) autonomy vs. shame and doubt (2 to 3 years), (3) initiative vs. guilt (4 to 5 years), (4) industry vs. inferiority (6 to 11 years), (5) identity vs. role diffusion (12 to 18 years), (6) intimacy vs. isolation (young childhood), (7) generativity vs. stagnation (middle adulthood), and (8) ego integrity vs. despair (later adulthood). Of particular interest with reference to the initiation of smoking are Erikson's fourth and fifth psychosocial crises.

Both the struggle to overcome inferiority and the effort to establish a self identity have been cited in one form or another by numerous researchers interested in interpreting the initiation of smoking in adolescents. For example, Erikson's "identity-crisis" in adolescence (being torn between the roles of child and adult) might be an interesting basis for explaining the apparent influence of peer pressure in the initiation of smoking, particularly if this notion were explored in some depth empirically.

A third contribution which has greatly influenced developmental and social psychology is Bandura's Social Learning Theory (*6*). Bandura's theory, which is concerned with imitative or modeling processes, would also seem to be useful in understanding the processes involved in the initiation of smoking. Social learning theory emphasizes the roles played by vicarious, symbolic, and self-regulatory processes in the acquisition of behavior. Further, this theory suggests the importance of reciprocal determination or the continuous mutual interaction between self-generated and environmental determinants in exploring human behavior. Bandura sees social learning as governed by four component processes: attention, retention, motor reproduction, and motivation or incentive.

Smoking appears to be initiated as a result of social influences or, more particularly, the imitation of models such as peers, media stereotypes, and significant adults (e.g., parents and teachers) (*27*). Considering the nature of smoking, a behavior with possible delayed aversive consequences and often more immediate social reinforcing consequences (especially for children and adolescents), it would seem that investigating smoking within the social learning paradigm would generate many useful hypotheses concerning the initiation of smoking. For example, the impact on children of the models of smoking parents or the impact of smoking adult models depicted in the mass media could be further explored in the context of social learning.

Communications models which examine information processing hold some promise for understanding the factors underlying the initiation of smoking as well as for developing more effective prevention programs. McGuire's (*53*) Communication Persuasion Model, for example, analyzes the persuasive impact of communications according to five component processes: attention, comprehension, yielding, retention, and action.

If the communicator wants the message to be accepted and acted upon, it is important to remember that individuals exposed to the message must be paying attention if communication is even to begin. Comprehension of the contents of the message is equally important. Yielding to or agreeing with the conclusions advocated in the message is vital if the communication is to have effects in the desired direction. Retention, or the maintenance of the induced agreement, is particularly important if the beliefs are to be operative when the individual is challenged by exposure to messages countering the accepted belief. By measuring the individual's response to such challenges, a useful evaluation of the impact of the communication on the subject, the degree of yield to the message, and the amount of resulting behavioral change or action resulting from the message may be obtained. McGuire's model would appear to be useful in both preparing and evaluating communications related to smoking prevention programs for children.

One of the most interesting aspects of McGuire's model is his "inoculation" approach to attitude change. McGuire suggests that existing attitudes may be strengthened by inoculating individuals against counter arguments to which they may be exposed. The application of this model to the pressures to initiate smoking would consist of "inoculating" adolescents against the social pressures to smoke which they may encounter at some future time. For example, Evans, et al. (*31*), using this approach in filmed messages, acquaint adolescents with the nature of the various social pressures to smoke. In a second film, they are inoculated against these pressures by being presented coping "strategies" based on information obtained from adolescents themselves. Further variations of such an inoculation approach would appear to be a promising means of relating a concept in social psychology to the deterrence of smoking in children and adolescents.

Typical Psychosocial Influences on the Smoking Decision

As mentioned earlier, despite extensive educational efforts, the onset of smoking in school-aged children continues relatively unabated, with age and grade level at which smoking begins reflecting a downward trend from high school and junior high school into the elementary grades (*61*). This trend has been reported consistently in the literature (*18*, *29*, *84*) and has grown at such an alarming rate that Kelson, et al. (*46*) refer to it as "the growing epidemic." It is generally agreed that the most effective way to attack the problem would be to influence children not to initiate smoking (*29*, *88*). Developing strategies of deterrence is dependent upon identifying those influences that lead children to begin smoking. While not all influences have been identified, many of them can be discerned in the literature related to children and smoking. Predictably, the influences most frequently cited include the role of the family, pressures from peer groups, formal education programs, and the effects of messages transmitted through the mass media. To a lesser extent, studies that explore the influences of individual differences and environmental factors have been reported.

Changing Sex Roles

As mentioned earlier, the disappearance of differences between the incidence of smoking of boys and girls is quite apparent (*61*). The reasons for these differences are not clearly established. Possible explanations, such as a differential impact of antismoking messages on the two sexes, have not yet been empirically demonstrated. Another possibility is that many social differences between the sexes are gradually disappearing in the light of the women's movement. A third possibility derives from the finding that smoking by teenage girls may have been perceived as more socially acceptable in 1974 than in 1968. This may have resulted in more honest self-reports of smoking; so instead of teenage girls actually smoking more, a more accurate indication of smoking by girls was being recorded.

Parental Smoking Habits

Parents who smoke clearly influence the smoking behavior of their children. In families where both parents smoke, 22.2 percent of the boys and 20.7 percent of the girls are also smokers, compared to 11.3 percent and 7.6 percent where neither parent smokes (*61*). These proportions have remained consistent over time. Merki (*55*) lists parental smoking habits as a major factor directly related to smoking by junior and senior high school students. Wohlford (*89*) uses identification theory to predict a direct relationship between parent and child smoking behavior. This relationship appears to be stronger for boys than for girls, a finding Wohlford attributes to stronger peer influences relative to smoking for girls. A recent American Cancer Society study (*58*) seems to confirm this notion. Borland and Rudolph (*9*) indicate that parental smoking is the second best predictor of smoking behavior in high school students. Palmer (*68*) reports similar findings for junior high school students. Edson (*23*) discusses both parental modeling and children's efforts to combat parental smoking as a result of the School Health Curriculum Project. Evans, et al. (*31*), in a smoking-deterrence investigation, incorporate a positive message for coping with parental smoking models, emphasizing that children can resist the pressure to imitate parents who smoke. Programs designed to educate parents who smoke on how they may be influencing their children to smoke should be considered important components of prevention programs. Also, research should be encouraged to examine the precise effects on the child of the smoking parent.

Parental Acceptance of Children's Smoking

While parental approval of smoking has been suggested as a contributing factor in influencing children to smoke, Allegrante, et al. (*3*) do not find parental approval to be a signficant factor, confirming Williams' (*88*) earlier conclusion that both smoking and nonsmoking junior high students report that their parents disapprove or would disapprove of their smoking.

Siblings Who Smoke

Although Piper, et al. (*70*) report no significant relationship between older siblings and the smoking behavior of the subjects in their longitudinal study, two major surveys (*61*, *88*) implicate the smoking behavior of older siblings as a possible influence on younger children. Twenty-eight to thirty percent of the boys and 25 to 26 percent of the girls who report regular smoking also have older siblings who smoke. If an older sibling and both parents smoke, the child is four times as likely to smoke as a child who has no smoking model in the family (*61*). Williams also reports the lowest incidence (4.2 percent) of smoking in those children who live in a household where neither parent smokes and where there are older siblings, none of whom smoke.

Rebellion Against Family Authority

While cigarette smoking as a form of rebellion against family and adult authority has not received much attention in the literature, a recent survey (*42*) indicates that smoking among teenage girls may reflect rebellious, anti-authority behavior.

Peer Pressures

Peer pressure is widely assumed to be a significant causal factor in the initiation of smoking. The strong influence of peer group pressures is generally evident in young adolescents (*38*, *78*), but the precise relationship of such pressure to the initiation of smoking is more difficult to establish.

In an intensive participant-observation study of ninth-grade students with a follow-up 2 years later, Newman (*64*) reports that peer pressure and conformity to group status norms were perceived by subjects to be major factors in smoking. The relationship was not as strong when the subjects were in the 11th grade, but was significantly different at both grade levels (*63*). A survey by Palmer (*68*) of more than 3,000 junior high school students finds that the prevailing peer model to be the single most important variable contributing to the onset of smoking in this age group.

In a longitudinal study of Canadian school children, Matthews (*51*) finds that peer influence was a major factor in the initiation of smoking in the population surveyed. The influence of peers seems to come from "best friend" relationships, rather than from large or diversified group pressure. In a multivariate study of correlative factors in youthful cigarette smoking, Levitt and Edwards (*50*) report that having a best friend or group of friends who smoke appears to be the best predictor of smoking in children from the 5th through the 12th grade. Bynner (*13*) finds the most important variable in explaining smoking behavior in English and Welsh schoolboys is the number of their friends who smoke. Williams (*88*) reviews a substantial number of studies which also conclude that pressures from peers and best friends are important influences to smoke.

In prevention programs, Newman (*63*) cautions against the utilization of nonsmoking student models whose general characteristics differ from those of the target population. The use of such models may alienate the target population against the antismoking message. Evans (*27, 31*) approaches the peer-pressure problem by presenting strategies for resisting peer pressure as filmed-sequence roles played by students selected from the target population.

School Environment

Specific school health education programs are addressed comprehensively in other chapters in this report. The dominant role of the school in the life of children and adolescents suggests the importance of the school environment in providing influences guiding the smoking decisions of children. Two important recommendations specified by the American Association for Health, Physical Education, and Recreation (*4*) are for schools to accept the responsibility for providing smoking education programs and for teachers and other school personnel to implement these programs.

The role of teachers, health professionals, and other adult role models as exemplars for the young is examined by a number of researchers (*16, 62, 80*). It may be important that such adult role models make positive statements related to their position on smoking. For example, teenagers perceive teachers as likely to be smokers (*42*). Sixty-eight percent of the girls and 67 percent of the boys judge most teachers to be smokers. A recent American Cancer Society survey (*5*) states that only 23 percent of female teachers and 18 percent of male teachers actually smoke. Such a difference in actual and perceived smoking behavior indicates a lack of communication in an area that could be critical in influencing the smoking decision in children and young adolescents.

Mass Media

In a Task Force Report on Respiratory Diseases, the National Institutes of Health (*60*) states that mass media have been used extensively in antismoking efforts, but exactly how they influence behavior is unclear. Ward (*87*) reports that, in a study designed to ascertain attitudes toward television commercials and to analyze the effects of television advertising on adolescents, the television medium appears to influence the formation of ideas and attitudes, yet does not "trigger" adolescents to buy a product. Ward's study indicates that cigarette ads are perceived by teenagers as hypocritical and are listed

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as "least-liked" while antismoking ads are perceived as "straightforward" and are liked. The effects of messages in other media, such as billboards, magazines, and displays need to be more precisely studied. Mendelsohn (*54*) concludes that, in general, current mass media efforts to educate the public concerning health issues are disappointing. It is possible that because of cognitive and social differences in various development stages of children and adolescents, mass communications may not be the most appropriate means to reach children and adolescents with smoking-deterrence messages. More specifically, targeted communications might be better presented in selected target situations.

Individual Characteristics

The notion of being able to identify potential smokers has been an elusive goal for researchers. There are very few investigations relating personality variables to teenage smoking. Smith's (*79*) review of 35 personality and smoking studies found only four related to teenage smoking. After a search of the literature related to personality variables that may influence the initiation of smoking, Williams (*88*) concludes that "both the empirical results of previous studies and discussions of the state of the art of research into personality correlates suggest that personality will not provide the most fruitful approach to understanding why children do or do not take up cigarette smoking" (p. 15). There appears to be some agreement that personality is more related to the amount smoked than to who will begin to smoke (*17, 52, 85*).

Individual differences in smoking are related to variables such as age-in-grade, achievement in areas important to the young person, social involvement, and participation in organized activities. Creswell, et al. (*18*), and Laoye, et al. (*48*) find that student educational expectations are related to their smoking behavior. Creswell, et al. (*18*) also find some support for a relationship between above average modal age and smoking behavior. They find smoking to be perceived as a compensatory behavior for students who had not achieved success in more traditional roles. Hasenfus (*37*) postulates that children and young people may begin smoking out of a normal curiosity, but soon come to view smoking as a coping behavior similar to adult usage. Bergin and Wake (*7*) state that teenage smoking appears to be triggered by changes in living habits such as changes in residence, absence of a parent, or matriculation in a university. No conceptual framework or organized line of research has systematically guided the research related to individual characteristics in the initiation of smoking, and the literature reflects the patchwork quality of the existing knowledge.

Perceptions of Dangers of Smoking

A recent trend in smoking and health research involves an attempt to identify and modify perceptions on the part of children and adolescents of the dangers of smoking. Evans, et al. (*29*) suggest that fear-based smoking-deterrence messages to this age group, enumerating the future costs of smoking—heart disease, lung cancer, and other serious diseases or death—are often ineffective because most children and young adolescents are more present- than future-oriented. They find it difficult to perceive such future dangers as meaningful or even important. Studies designed to communicate the immediate physiological effects of cigarette smoking on healthy young people (*35, 77*) may help to make the health dangers more immediate and compelling. Filmed demonstrations comparing teenage smokers and nonsmokers by the nicotine in their saliva, the carbon monoxide in their breath, and their heart function are components of the 3-year longitudinal study by Evans, et al. (*31*).

Critical Evaluations of Some Current Prevention Programs

Several reviewers (*29, 34, 67*) point out the serious limitations that exist in evaluating research in this area. A lack of common definitions of smoking behavior, reliance on self-reporting and lack of objective measures of smoking, attrition rates in long-term studies, inappropriate statistical analyses, biased sampling errors inherent in using available volunteer populations, and lack of appropriate control groups are major limitations of the vast majority of the studies reviewed. The results of such studies must thus be viewed with caution.

Most smoking prevention programs have not been specifically directed at children and adolescents who logically should be the key target of such programs. Rather, they have been general public information campaigns conducted by private and governmental agencies, such as the American Heart Association, the American Cancer Society, and the U.S. Public Health Service. Various in-school educational programs incorporating information concerning the health hazards of smoking into course curricula and special programs with certain unique features have also been instituted.

Public Information Campaigns

Major criticisms are leveled at many public information smoking-prevention campaigns. Too often these programs fail to build in adequate evaluations. Also, they tend to be notional and atheoretical. Content and persuasive strategies in these campaigns are too often arbitrarily chosen, based on subjective judgment, rather than being systematically pretested. Bradshaw (*11*) reviews 14 public educational campaigns between 1960 and 1970 involving local communities, schools, and universities in both the United States and the United Kingdom. He concludes that the effects of these campaigns on smoking behavior have been minimal at best with many producing no apparent effect. The failure to conduct adequate follow-up evaluations and to include comparison control groups in studies carried out are among other criticisms made of these campaigns. Recognizing the many limitations of these campaigns, Bradshaw calls for more systematically developed communications which can become the basis of widely disseminated programs to deter young people from acquiring the smoking habit.

Public information campaigns aimed at prevention can also be criticized for failing to evaluate the program's impact over extended periods of time. For example, Fishbein (*34*), in a recent report to the Federal Trade Commission, indicates that at the present time we do not have enough information about the beliefs, attitudes, and intentions already held by the public with respect to smoking decisions (i.e., to initiate, reduce, increase, or stop) or information regarding the degree to which these decisions are under attitudinal or normative control. Fishbein suggests that this information is necessary in order to develop communication materials of all kinds that would contain the most appropriate arguments for affecting a given smoking decision. Concluding his report, he states that "Although there is much that could be done immediately to inform the public, much more research is necessary if one wishes to maximize the likelihood that information will also influence a smoking decision" (p. vi).

Most critically, public information campaigns directed at prevention of smoking have been too broadly targeted. They have not reflected the beliefs, attitudes, and intentions held by what should be the prime target for prevention programs: children and adolescents. As mentioned earlier, such campaigns must take into consideration the specific developmental level of the child or adolescent. Evans, et al. (*31*), for example, find that older adolescents may respond to different smoking prevention messages than younger adolescents.

School Programs

The majority of school programs are preventive in intent, whether they are oriented toward exploring generic research issues or are merely single classroom demonstrations of so called "hands-on" programs designed to illustrate some specific aspect of smoking.

Unfortunately, the vast majority of such programs possess methodological shortcomings, particularly in evaluation designs. Many of the reports of these programs fail to present the documentation necessary for the most rudimentary evaluation by the reader. It should be noted, however, that much of the literature related to children and smoking is found in publications that may not require or encourage reports which are carefully detailed and which include rigorous evaluations.

Many of these reports are anecdotal or descriptive in nature or are offered merely as guidelines for curriculum planning and implementation. Such a morass of programs reported so loosely cannot be compared within any theoretical framework. This leads to frequent repetition of efforts. It appears that in school smoking-prevention programs, the "wheel" is regularly reinvented. Since a critical evaluation of most school programs is thus virtually impossible, at least some observations concerning current school programs will be presented and the implications of these observations for planning more rigorously evaluated programs will be discussed.

In a recent review, Thompson (*84*) expresses a general cynicism concerning the effectiveness of school programs. She further states that multimethod campaigns and youth-to-youth programs are generally ineffective. Terry and Woodward (*82*) report that relatively few teachers are trained as health educators, and Chen and Rakip (*15*) find serious problems in teacher implementation of programs on smoking and health. Teachers themselves often express a lack of confidence in their ability effectively to implement smoking education programs. This inability may be reflected in Levitt's (*49*) survey of 50,000 Indiana school children, in which less than 1 percent of the students indicate receiving information about smoking in school health classes. A comprehensive program for teacher training, at the preservice and inservice levels, in evaluating and implementing smoking and health programs is an area where effective action could be taken based on present knowledge and research.

One promising trend involves preplanned longitudinal, comprehensive studies in school settings carried out by large institutions (e.g., universities) with a strong commitment to evaluation. The pressure to produce immediate and specific effects on smoking is somewhat lessened because they are being carried out in the context of long-range evaluation. Thus the investigator has the opportunity to design conceptually sound projects based on sophisticated models. Such studies are also fruitful in producing spinoff studies that test specific hypotheses, pinpoint effects, and eliminate unworkable approaches. Stringent preplanned evaluation is an integral part of the best of these in-school programs. While such long range programs, implemented and evaluated over substantial periods of time, are both costly and difficult to manage scientifically and logistically, the data produced may have important implications for developing systematic theoretical concepts and in generating new research. Such studies may come closer to isolating the complex social, physiological, and psychological factors that underlie the smoking phenomenon. Generally, such programs are carried out so that the community continues to benefit from the program after its completion, since it provides pretested and evaluated materials for incorporation into school curricula.

One of the best known of the longitudinal, comprehensive studies is the National Clearinghouse for Smoking and Health's School Health Curriculum Project (based on the so-called Berkeley model) that has been introduced into more than 200 school districts in 28 States. The curriculum is based on results of empirically tested concepts related to communicating health knowledge to children, including information about smoking. It is being implemented in programs from kindergarten through seventh grade at the present time. Evaluation components of the program are just now beginning to yield results. In the smoking area, a substantial relationship between enrollment and nonenrollment in the program and smoking knowledge and behavior has been claimed (*58*). However, a careful inspection of the quasi-experimental study on which that assertion is based reveals only small inconsistent differences (*56*). Detailed descriptions of the implementation of this program are given by Edson (*23*), Caramanica, et al. (*14*), and Albino and Davis (*2*). (The School Health Curriculum Project is discussed more fully in another chapter in this report.)

The University of Illinois Antismoking Education Study (*19, 20*) has been underway for more than a decade. It has produced several smoking-measurement instruments that have been used in a number of smoking studies. These instruments incorporate informational, attitudinal, and self-report behavioral components but have not been validated against more objective measures of actual smoking.

The Illinois Antismoking Education Study generated several kinds of studies which address themselves to evaluating various in-school approaches to control smoking. For example, in one study, Irwin, et al. (*41*) examine the relative impact of the regular classroom teacher as a smoking information communicator compared with teachers especially trained in health communication. Although they find that the classroom teacher was at least as effective as the specially trained teacher, more recent studies (*82*) do not necessarily support this conclusion. An intention-to-smoke measure was also developed as a result of the Illinois study. Using this measure, Laoye, et al. (*48*) find that a 2-year projection of smoking could be successfully demonstrated. Merki, et al. (*55*) explore smoking behavior of rural high school students and find that student smoking is related to parental smoking habits, participation in school group activities, and lower educational aspirations. From a 9-month participant-observation study, Newman (*63, 64*) concludes that both covert and overt smoking are low-status activities for ninth grade girls and overt smoking is a low-status activity for boys. (The Illinois study is also described more fully elsewhere in another chapter in this report.)

In Houston a 3-year longitudinal study reported by Evans, et al. (*31*) is being undertaken. It is designed to train junior high school students to resist the pressures to smoke from peers, the media, and models of smoking parents. Also involved in this study are interventions that monitor smoking and those that communicate immediate physiological effects of smoking. A nicotine-in-saliva measure is employed to increase the validity of self-reports of smoking. A major purpose of the study is to explore the feasibility of incorporating into school health programs inoculations-against-social-pressures-to-smoke messages in lieu of the frequently used fear-arousal, impersonal, information-centered communications. Preliminary results indicate that such intervention strategies, based on the use of films whose content is derived from feedback from students themselves, may be effective with some students in deterring the onset of addicted smoking, although the final results await the completion of the final years of the investigation. Also, further replications of this general approach to thwarting smoking behavior in adolescents, using either films or more personalized interventions, are being undertaken at Stanford (Cheryl Perry), the University of Minnesota (C. A. Johnson), Tyler, Texas (Richard Evans), and elsewhere.

General Comments

Obviously, the psychosocial factors that influence the initiation of smoking are varied and complex. Aside from a few promising prevention programs, most of them fail to encompass psychosocial conceptual frameworks. Obviously, there is also a great need for such programs to be more carefully planned, controlled, and evaluated.

Fodor, et al. (*36*) propose that educational programs that deal with the totality of man as a complex being offer the most promise. "Smoking education must, in fact, become health education, taking into consideration the multiplicity of factors related to smoking and health—physical, mental, and social" (p. 94). Rabinowitz and Zimmerli (*72*) recognize the complex, long-range problem:

> What seems most crucial for future health education planning.....is that a 'one-size-fits-all' approach is contraindicated to student health teaching in terms of message content, structure, and perhaps, classroom delivery. To achieve comparable outcomes it may be essential that several distinct approaches to smoking education be explored for social subgroups with demonstrably different backgrounds of exposure, involvement, and maturation (p. 330).

The best efforts at present appear to possess at least some conceptual basis, are long-term, multiphasic studies attempting to establish good baseline data, develop and test specific hypotheses using carefully controlled methods of investigation, employ objective measures of smoking to validate self-reports, and include evaluations of the program through several years of implementation.

The ideal prevention program would follow the example of Sweden (*76*) where a 25-year effort has begun whose objective is to make those born in 1975 a nonsmoking generation. The program began in 1974 with expectant parents and is presently concentrating on withdrawal clinics and other measures to develop a nonsmoking environment for those children born in 1975. Educational efforts for adults and children and increased governmental control over advertising and marketing of tobacco products are being implemented, and an all-out effort is being made to create a nonsmoking generation in a nonsmoking environment, supported by both governmental efforts and the general public.

Some Recommendations for Future Research and Prevention Programs

Although recommendations for future research and prevention programs logically emerged in several earlier sections of this chapter, some additional recommendations may be in order. Most of the current research concerning psychosocial determinants of smoking in children and adolescents tends to be correlational in nature. Because of the limited amount of variance accounted for, it is difficult to establish a precise linkage between any given psychosocial influence and the initiation of smoking. Just as Jessor and Jessor (*43*) have found with respect to the use of other drugs, it is likely that an array of social influences precipitates the onset of smoking. What may be needed now is the selection of some of these specific influences for particular attention. For example, the influence of the mass media on smoking initiation, which currently appears to be uncertain, might be better understood through a series of small, well-controlled basic investigations. The results of such investigations should be interpreted within the context of the broader impact of the mass media on the behavior of children and adolescents to avoid the criticisms leveled at how the research concerning violence and television was conducted. Additionally, just as the focus in the area of television or films and behavior has shifted from exploring how they precipitate antisocial behavior to how they may encourage prosocial behavior (*6*), some of these investigations should also examine how the mass media have perhaps inadvertently contributed to the child's decision *not* to begin smoking, or to quit before he or she has become a confirmed smoker. Perhaps the use of mass media to counter prosmoking influences should also be further explored. A similar approach might be used to explore more explicitly how to counteract the impact of social pressures in the initiation of smoking (*27*, *31*).

Lacking in most of the investigations reviewed is an adequate conceptual base. As discussed earlier, certain types of major conceptual models in developmental and social psychology have gone virtually unexplored as a source of hypotheses for research in the area of smoking in children and adolescents. Many other current conceptual directions in psychology could well be explored as they relate to smoking. The theory of cognitive dissonance (*33*), Fishbein's belief-behavior concepts (*34*), Kohlberg's theory of moral development (*47*), impression formation (*81*), attribution theory (*44*, *45*), decision-making in children (*12*), Jessor and Jessor's multi-determinant conceptual structure of problem behavior (*43*), and the concept of risk-taking (*21*) are all examples of theoretical areas that might generate some testable hypotheses in this area of smoking.

Still another important area of research would be to explore the interrelationship of the initiation of smoking in children with other health behaviors. For example, some provocative studies (*8, 40*), though not confirmed by other studies such as O'Donnell's (*66*), suggest that smoking may be a "drug entrance ticket." Children who begin smoking are more likely to begin using alcohol and hard narcotics. Certainly, a careful examination of such types of health-behavioral interrelationships would be a crucial area of research. Likewise, how does smoking relate to the over-all lifestyle of the developing child? A look at the "natural development" of the smoker, perhaps even completing a few studies, such as those the Jessors (*43*) have done with drug usage, which examine very small samples of children over time, might generate a number of significant hypotheses.

However, as is being demonstrated in at least one current investigation (*31*), useful intervention programs might already be developed which may have a better chance of having a long-term impact on the smoking behavior of adolescents than the largely fear-arousal, impersonal, information-oriented approaches generally used. Virtually all investigations in this area report that adolescent smokers and nonsmokers alike really believe that smoking is potentially dangerous to one's health (*34*). Obviously, this fear does not appear to be enough to deter the onset of smoking or to be sufficiently successful in motivating smokers to stop (*31*). Therefore, other types of emphases in prevention programs should be developed. Such intervention programs should apply the method of successive approximation. At each step of the way, the target population of children or adolescents should provide input into the content of the intervention within the context of an appropriate psychosocial, conceptual framework. All intervention materials should be pretested on the children.

Whatever the content of the intervention program, great care should be taken to plan and utilize an adequate evaluation methodology. Failure to incorporate rigorous evaluation procedures emerges as a significant limitation of virtually all of the intervention programs reviewed. One particularly troublesome problem in evaluation methodology deals with the appropriate criterion for the impact of a program. Measures of information about smoking, attitudes towards smoking, or self-reports of smoking may not be adequate indicators of a program's impact. Serious questions are raised in contemporary social psychological literature (*30, 32*) concerning the relationship between information gain and attitude change and behavior. It would be most unfortunate to conclude that a demonstration of the presence of increased information about smoking dangers or an attitude change toward smoking has necessarily had a significant impact on smoking behavior.

Furthermore, as smoking among children and young adolescents is a taboo and socially unacceptable behavior in many social settings (e.g., in schools), self-reports of smoking may be inaccurate.

The majority of the investigations reviewed, whether they are examinations of psychosocial factors, surveys, smoking informational campaigns, or in-school educational programs, rely heavily upon self-report measures of smoking. Investigators (*73*) in the behavioral science literature describe the existence of an acquiescience or interpersonal expectation effect; that is, subjects report what they believe the experimenter expects whether or not it is a true reflection of their actual behavior. Dunn (*22*) questions how much credence can be given to the introspective reports of smokers. He states: "Factors such as the need for social approval of opinions and actions, the need to justify a preference commitment, order of presentation effects, brand imagery effects, halo effects, and the yea-saying tendency are collectively more determinative of a report of a smoke-induced sensory experience than is the sensory experience itself" (p. 98). Although this statement refers principally to self-reports of motivational factors in smoking, many of the same points can be applied to questioning the validity of self-reports of smoking itself.

Obviously, measures of smoking behavior that are more objective than self-reports of smoking are vital for a valid evaluation of programmed treatments. One such measure has been reported (*28, 31*). This involves the use of a procedure which appears to increase the validity of self-reports of smoking behavior. A mass spectrometric analysis of nicotine-in-saliva (*39*) is used to increase the validity of self-reports. Films depicting this analysis procedure are shown to students before they have produced a saliva specimen and before they are requested to record self-reports of their smoking behavior. This results in significantly more reports of smoking. Other investigators (*74*) are exploring the use of chemical indicators of smoking. However, using only direct chemical indicators as the major dependent measures may be too costly or may only be recording recent smoking. For example, nicotine, because of its "half-life" when measured in the blood, records smoking for only a very brief period (*28*). Developing improved techniques for more direct measurement of smoking is clearly an important area for future investigations.

Finally, future research and prevention programs should address themselves to the problem of establishing a truly long-term impact. Many smoking prevention programs often report optimistic success rates. The reporting of such success rates should be qualified by the possibility of the individual beginning to smoke at some later time. Inferences about the evolution of smoking suggest that by the end of the ninth grade very few adolescents are confirmed smokers. The critical level of the onset of confirmed smoking appears to be in high school (*88*). Therefore, the true impact of any deterrence-of-smoking program with adolescents may not even be measurable until after the adolescent has entered high school. This problem is not unlike the backsliding or recidivism encountered in virtually all smoking cessation programs (*71, 83*).

Thus, in recommendations for future research and in the development and implementation of prevention programs with children and adolescents, the range of possibilities appears vast. Perhaps with a focus on the initiation of smoking, much critical new knowledge of the developing life style of children and adolescents will also emerge. Surely, smoking must be regarded within the total context of the individual's development. Perhaps the real question to be answered is: why do we knowingly choose to engage in self-destructive behavior when so much of our energy is directed toward preserving our lives?

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Psychosocial Influences on Cigarette Smoking

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Maintenance of Smoking

Many of the psychosocial influences on the establishment of smoking are discussed at length in other chapters of this report. This chapter begins with issues related to the maintenance of cigarette smoking. Much of the research which was reviewed, however, made no strict distinction between factors leading to the establishment and those leading to the maintenance of smoking. For a more far-ranging review than possible in this short space and for a somewhat different approach to the topic, the reader is advised to consult other sources (e.g. *47*, *48*).

Individual Factors

Personality and Smoking

In part because such research can be among the easiest to conduct, many studies have been undertaken to correlate scores on self-report personality inventories with smoking habits. Much of this research has been marred by too few subjects, inadequate samples, too little attention to other measurable and potent influences on cigarette smoking, such as peer pressure, parental influence, and socioeconomic status, and too little appreciation of the fact that studying the determinants of cigarette smoking is fundamentally a problem for multivariate analysis (see the criticisms in *19*, *22*, *49*, *65*, *90*).

In general, the personality research shows that even the most reliable personality predictors of cigarette smoking, such as extraversion, account for only about 3 to 5 percent of the variance in measures of smoking habits. Smith (*90*) concludes that the best univariate personality assessments are able to discriminate smokers from nonsmokers in only about 60 percent of the cases. His own multivariate studies are able to discriminate smokers from nonsmokers in 68 to 76 percent of the cases.

Personality research is intrinsically correlational. It describes associations between variables and does not establish causal connections. Researchers are in a position to manipulate at random (a requirement for true experimental designs) neither the personalities nor the chronic smoking habits of their subjects. To find that smokers are, to use the same example, more extraverted than nonsmokers gives no information about (1) whether smoking caused an increase in extraversion, or extraversion caused an increase in smoking, or (2) whether some unmeasured confounding variables, which are correlated with both smoking and extraversion, are the true cause of the observed association. Longitudinal studies that are able to assess personality before the onset of smoking are some help in dealing with the first problem, but they deal not at all with the second. Even with these limitations in mind, the search for correlations between personality and smoking has yielded some information worthy of consideration.

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Wiggins (*105*) reviews studies which indicate that most of the various measures of temperament can be boiled down to two major factors—extraversion and neuroticism (anxiety).

Extraversion

Since the first major review of this area by Matarazzo and Saslow (*54*), a cluster of variables often called extraversion has been shown to be positively associated with cigarette smoking. Eysenck's work on extraversion-introversion has had a powerful influence on defining the field (*27*). According to his research, the typical extravert craves excitement, is willing to take risks, is sociable, likes parties, is carefree and easygoing, and may be aggressive. On the other hand, the introvert is introspective, retiring, bookish, prudent, emotionally-controlled, passive, and reliable. Eysenck considers the extraversion-introversion dimension to be comprised of varying degrees of four major traits: sociability, liveliness, impulsiveness, and jocularity. In a carefully sampled study (*28*), which also controlled for age and social class in British males, the amount smoked was related directly to greater extraversion.

Cattell's work with his 16PF inventory on a sample of college men and women (*14*) supports this finding on extraversion. Extraversion emerges as a second-order factor of the 16PF and correlates +.21 with smoking (a three-point scale of smoking habits). The primary factors which correlate most with smoking are Affectothymia (outgoing) ($r = +.16$) and Surgency (happy-go-lucky) ($r = +.29$). Both these factors are major components of the extraversion scores.

Smith (*91*) reviews the results of 15 reports describing 25 studies that he believes have provided adequate measures of extraversion (e.g., the Maudsley Personality Inventory, MMPI Social Introversion Scale, 16PF: Extraversion, Strong Vocational Interest Blank, and peer ratings of extraversion). Twenty-two of the twenty-four studies that describe statistical analyses showed that smokers were more extraverted than nonsmokers. It was noted that the effect has been found in several different populations (for example, U.S. adult males and females, British adult males, U.S. high school and junior high school males and females). Smith (*91*) treats impulsiveness as a separate personality category. But perhaps it is best to consider the impulsiveness findings as part of the general trend for smokers to be more extraverted. It has been argued that there are two basic components of extraversion: sociability and impulsiveness. Eysenck (*28*), for example, demonstrates that neither factor alone contributes inordinately to the association between smoking and extraversion.

More recent research (*15, 18, 69*) in general supports the association between smoking and extraversion. The Cherry and Kiernan paper (*15*) is of special interest because it describes the results of a large sample, longitudinal study. Personality scores were obtained on the Maudsley

Personality Inventory at the age of 16 years. (Neuroticism findings will be discussed below.) Smoking habits were measured when subjects were 25 years old. The total usable sample was 2,753 British males and females. Both male and female smokers were more extraverted than male and female nonsmokers ($p < .01$). An analysis of recruitment to smoking in those who had not been regular smokers by their 17th birthday showed that extraversion, neuroticism, and being male were each independently and positively associated with becoming a smoker. (There was an indication of interaction between the neuroticism and extraversion effects; those high in both were less likely to be smokers than would have been predicted.)

Russell (*73*) proposes that the following findings cluster with a degree of extraversion—that smokers are greater risk-takers, more impulsive, more prone to divorce and job changing, more interested in sex, and more likely to drink tea, coffee and alcohol.

Eysenck (*26*) has offered a biologically based theory as to why smoking should be more rewarding to extraverts than to introverts. Little additional social-psychological research has been done on how being extraverted might lead one to start or maintain smoking or on how being introverted might lead to not smoking. Likely hypotheses are easy to formulate. Since peer and parental pressures can be powerful influences on recruitment to smoking, it is interesting to note that extraverts are known to be more susceptible to social influence. Perhaps introverts are as resistant to social pressures to smoke as extraverts are prey to them. No research has been performed which attempts to hold these powerful social pressures constant to see the "purer" influence of extraversion on smoking. For example, the association between onset of smoking and extraversion may be moderated by some critical social variable. Future research should consider testing specific hypotheses about how extraversion and smoking could be related causally.

Neuroticism

Smith's review (*91*) uses the label "mental health" to loosely unite research that has gone under the more specialized labels of "neuroticism," "nervousness," "psychosomatic distress," "adjustment," "emotionality," and "anxiety." Just over half of the 50 or so studies in his review show smokers to have slightly poorer mental health than nonsmokers; the remaining studies show no relationship between smoking and neuroticism. The diversity of measures used and the lack of precise, consistent conceptualizations in this area may be responsible for much of the inconsistency. And it should be emphasized that the positive findings can in no way be interpreted to support the notion that smokers are substantially more neurotic, psychotic, or "crazy" than nonsmokers. At best, the data show a modest relationship between neuroticism and smoking, accounting for 1 or 2 percent of the variance.

Matarazzo and Saslow (*54*) report that for the most part smokers have higher neuroticism scores. The first Surgeon General's Report on Smoking and Health (*98*) concluded tentatively that smoking and neuroticism were probably related. Eysenck (*27*, *28*) has found no evidence that smokers are more neurotic in large representative samples of British adult males.

Two careful studies suggest that there may be sex differences in the relationship between smoking and neuroticism. Waters (*101*), in a random sample of 2,000 electors in Great Britain, was able to get completed questionnaires from 773 men and 945 women. For men, the correlation between smoking habits and neuroticism was essentially zero (Spearman's rank-order correlation coefficient between neurotic score and amount smoked was -.002); for women, the correlation was small, but statistically significant ($r = .127$, $p < .001$). Clausen (*17*), as part of the Oakland Growth Study, reports scores on psychoneurotic symptoms for boys and girls who would later grow up to be smokers. Males show a generally negative relationship between amount smoked during adulthood and their adolescent neuroticism scores; females show a generally positive association between smoking and neuroticism.

One other major British survey study, using a short form of the Maudsley Personality Inventory, finds no significant trend for neuroticism to increase among smokers as the amount smoked increased, but does find some indication that such a trend was present for women (*15*); when a simple nonsmoker-smoker classification was used, neuroticism was higher in both male and female respondents. In Indian males, who smoked either 0, 1 to 10, 11 to 20, or over 21 cigarettes per day, neuroticism decreased as smoking increased. Both linear and cubic trend were significant statistically (*43*).

In a detailed study on smoking and habits of nervous tension, Thomas (*96*) surveyed male medical students at Johns Hopkins University (437 nonsmokers, 144 ex-smokers, 251 continuing cigarette smokers) and found an anxiety scale significantly related to greater smoking in a stepwise discriminant function analysis.

At present, the most reasonable conclusion concerning smoking and neuroticism is that there are systematic relationships between them. Researchers do not yet understand, however, the interacting variables or moderating influences on the relationship. It is interesting to note here that Lebovits, et al. (*50*) evaluated the effects of defensiveness, age, education, and smoking habits on the MMPI scores of 1,572 white males, aged 40 to 56; they looked for statistical interactions which influenced the scores and found indications of some small interactive effects. More research along these lines might reveal the boundary

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conditions that influence the relationship between neuroticism and smoking.

Some authorities, e.g., Russell (*73*), have proposed that slight neuroticism may be the result of being a dependent cigarette smoker rather than a cause of smoking; cigarette withdrawal syndromes may result in greater neuroticism. More careful evaluation of the characteristics of the individual's smoking habit—in particular, whether or not he or she is an *addicted* smoker—may help answer this question.

Antisocial Tendencies

Smith (*91*) considered 19 reports; 20 of 32 analyses showed that smokers had greater antisocial tendencies (belligerence, psychopathic deviance, misconduct, rebelliousness, defiance, and disagreeableness). Subsequent studies have supported this relationship (*49*, *62*, *69*).

Matarazzo and Saslow (*54*) and Weatherley (*102*) consider that smokers' greater antisocial tendencies may be due to a response bias. Perhaps smokers are more willing than nonsmokers to admit negative characteristics about themselves (*25*, *84*), even though in actuality they may not differ from nonsmokers in these characteristics. Smith argues that ratings by peers support the belief that smokers have greater antisocial tendencies and that, therefore, the response bias explanation is not very persuasive.

Internal-External Control

At the time of Smith's review (*90*), there had been only five tests of the relationship between smoking and internal-external control. Internally-controlled individuals tend to believe that they are the masters of what happens to them; their effort and skills (intrinsic properties) will bring them rewards. Externally-controlled individuals tend to believe that fate, luck, or, in general, things beyond their control will bring them their rewards. Four out of five analyses showed smokers to be more externally controlled. (The disconfirming analysis revealed a probability level of about .06, rather than the standard $p < .05$.) Two more recent studies (*5*, *36*) are divided in their support of the hypothesis that smokers are more externally controlled.

Miscellaneous Personality Variables

Orality has not been demonstrated conclusively to be related to more smoking (*91*). In addition, the concept of orality and its measurement are far from clear-cut. Some of the questionnaires intended to measure orality have depended on questions on beer drinking, coffee drinking, and medicine taking; hence, other drug use behaviors are being defined as "oral behaviors" (*40*).

The Edwards Personal Preference Schedule (EPPS) has shown some fairly consistent smoker-nonsmoker differences. Smokers tend to be higher in "heterosexuality" and lower in "deference" and "order" (*89, 90*).

Personality and Attitudes Toward Drug Taking

Stokes (*94*) has argued that traditional personality constructs are likely to be inadequate to the task of finding strong predictors of drug use and that personality-attitude measures should be more tailored to the issues of drug use. Six personality factors were tested: fear of personal reaction to drugs; dissatisfaction and a desire to change oneself; respect for the illegality of psychedelic drug use; sensual hedonism; philosophical hedonism; and general tendency to try drugs. The two most important predictors of tobacco use were "general tendency to use drugs" ($r(735) = .29$, $p < .001$) and "fear of personal reaction to drugs" ($r = .26$, $p < .001$). In a multiple regression analysis, the multiple R of the six factors with tobacco use was .349, accounting for 12 percent of the variance. It should be kept in mind, however, that as questionnaires themselves become more targeted on drug use and less on general personality structure, the nature of the research is altered.

Smoking Typologies

The most common strategy for discovering why people smoke has been simply to ask them on a questionnaire to indicate their agreement with statements on reasons for smoking (e.g., "I smoke cigarettes to stimulate me, to perk myself up") or on occasions for smoking (e.g., "I like to smoke when at a party"). Ikard, et al. (*38*)—employing a theoretical analysis by Tomkins (*97*)—factor-analyzed responses to proposed reasons for smoking. This analysis revealed six factors: Habitual (e.g., "I smoke cigarettes automatically without being aware of it"), Addictive (e.g., "Between cigarettes I get a craving that *only* a cigarette will satisfy"), Reduction of Negative Affect (e.g., "When I feel 'blue' or want to take my mind off cares and worries, I smoke cigarettes"), Pleasurable Relaxation (e.g., "Smoking cigarettes is pleasant and relaxing"), Stimulation (e.g., "I smoke cigarettes to give me a 'lift' "), and Sensorimotor Manipulation (e.g., "Part of the enjoyment of smoking ... comes from the steps I take to light up"). For both men and women, moderate correlations were found between average number of cigarettes smoked per day and the Habitual, Addictive, and Negative Affect Reduction factor scores. Although second-order factors are not reported, inspection of the intercorrelation matrix for the scores on the six types of smoking discloses correlations ranging from .38 and .58 among the Habitual, Addictive, and Negative Affect Reduction scales.

McKennell (*58*) replicated his earlier work and the work of Horn and his associates. In both cases, the factor structures were remarkably stable. The only revision warranted was the addition of an eighth factor to his own system—Reluctant Smoking. Reluctant Smoking was seen as similar to Horn's Habitual Smoking. In comparing the models, McKennell found that Horn's Pleasurable Relaxation was not measuring the same thing as was his own Relaxation Smoking. The Horn factor concerns smokers' general attitude toward smoking, that is, how pleasurable it is to smoke, while the McKennell factor concerns the desire to smoke in relaxed situations. The respective factors, Reduction of Negative Affect and Nervous Irritation Smoking, were found to be equivalent. McKennell concluded that it is possible to integrate the two models into a six-factors scheme. The first three factors load on a dimension of Inner Need (Inner Need/Relaxation, Inner Need/Stimulation, and Habit), the next two factors are concerned more with the sensorimotor and social aspects of smoking. The last and most tentative factor derives from Horn's Pleasurable Relaxation factor.

McKennell (*58*) used cluster analysis to determine if scores on these six integrated factors could be used to classify a random sample of 2,000 British respondents into distinct smoking types.

Six types were found(*58*, p. 10):

1. *Low Need-Pleasure* smokers, accounting for 14 percent of all smokers, tend more than others to be light smokers, with nonmanual occupations, who go to church, whose friends do not smoke, and who would not find it difficult to stop smoking.
2. *Medium Need* smokers, accounting for 30 percent of all smokers, differ from Low Need-Pleasure smokers chiefly in having a much more favourable attitude to smoking. Otherwise they are similar, although a little nearer the average in amount smoked.
3. *Medium Need/Handling-Social Confidence* smokers are a small group, comprising only 5 percent of all smokers. Apart from their motives for smoking, their most distinctive trait is their above-average frequency of drinking beer.
4. *Medium Need/Reluctant* smokers account for 28 percent of all smokers. They tend to disapprove of smoking but to be unable to escape from dependence on it. They tend to be young.
5. *High Need* smokers, who account for only 8 percent of all smokers, are distinct from High Need-Social smokers in scoring lower on the Handling and Social factors. In other respects they are similar.
6. *High Need-Social* smokers account for 15 percent of all smokers. They tend to smoke heavily, to have a manual occupation, to have friends who smoke, and to find it very difficult to stop smoking.

Coan (*18*) factor-analyzed an expanded version of the Horn scale and arrived at a classification scheme that is, in the main, compatible with the integration proposed by McKennell. Russell, et al. (*76*) compared the Horn and McKennell typologies, added new questions to their self-report inventories, and attempted to develop a typology that was more informed by recent developments in the psychopharmacology and social psychology of cigarette smoking. Six oblique factors were obtained: Psychosocial Smoking, Indulgent Smoking, Sensorimotor Smoking, Stimulation Smoking, Addictive Smoking, and Automatic Smoking. One of the most provocative findings of this analysis was that Horn's Negative Affect Reduction factor did not appear on its own, but was split between the Addictive and Stimulation factors. What McKennell had been describing as a second-order "inner need" factor is here called Pharmacological Addiction and is comprised of the stimulation, automatic, and addictive factors. (The correlations among these factors ranged from .50 to .63). Scores on these three factors were able to discriminate the primary sample of 175 cigarette smokers from a second group of 103 addicted heavy smokers who were attending smoking treatment clinics. The authors propose that the single dimension of pharmacological addiction to nicotine may prove more important for significant classifications of cigarette smokers than would profiles based on the six types of smoking. Perhaps cluster analyses as in McKennell (*58*) would help answer this question.

Smoking typologies based on what smokers can tell us about their reasons and occasions for smoking are, until proven otherwise, of limited value. It is unclear what insights these verbal reports give us into smoking behavior. Recent work in psychology questions seriously the validity of any self-reports of motivation (*64*). It is also clear that processes at work well beneath the level of awareness can influence cigarette consumption (*83*, *84*). A recent somewhat preliminary laboratory study indicates that there may be little behavioral validity to the self-reports about reasons for smoking; the classification of smokers into Positive Affect, Negative Affect, and Social Stimulation smokers did not relate to actual smoking behavior in various experimental conditions designed to elicit these types of smoking (*2*). Other research (*51*) suggests tentatively that verbal reports of reasons for smoking are more accurate for factors related to external cues (e.g., Pleasure-Taste and Habit) and less accurate for reports of internally defined states (Addiction).

Russell's (*74*) model of smoking proposes a progression from smoking for nonpharmacological rewards (that is, psychosocial and sensorimotor) to smoking to gain a positive effect from nicotine (indulgent, sedative, stimulation smoking). Finally, an addiction to nicotine develops and avoidance of the ill effects of nicotine withdrawal becomes an additional reinforcer of smoking.

It should be noted that Schwartz (*87*), using cluster analysis, detected 10 smoker types based on socioeconomic status, alcohol consumption-smoking environment, confidence-security adjustment, illness-anxiety, and attitudes toward smoking-beliefs about dangers. However, this result is not reported in enough detail so that it can be commented on at length.

The development of valid classification schemes for types of cigarette smoking could be a great boon to research on psychosocial influences on smoking. Perhaps, for example, the personality structure of addicted smokers is different from that of social smokers. Coan has conducted an interesting study which pursues this idea (*18*). Some greater standardization of behavioral classification of smoking habits is also advised. Clearly, a simple division of subjects into the categories of smoker versus nonsmoker is no longer excusable (*17*). Number of cigarettes smoked per day, number of months or years having been a smoker, nicotine content of preferred brands, and information about inhaling should be determined. (Eysenck (*28*) found that inhalers had a higher degree of neuroticism than those smokers who did not inhale.)

Self-reports of number of cigarettes consumed present their own problems of interpretation. First, there are strong pressures for the respondents to round-off their answers by saying "half a pack," "a pack," "pack and a half" and so on. Schachter has argued that, depending on the cut-off points that researchers use to establish their smoking categories, it is possible to arrive at some mistaken conclusions about the correlates of amount smoked (*82*). Using numbers of cigarettes smoked as the main indication of heavy or addicted smoking has had only modest success (*35, 38, 58, 76*). Another simple question promises to provide a surer link between addicted smoking and self-reports of the smoking habit—the time of the first cigarette in the morning. Kozlowski (*45*) and Schachter (*81*) have begun exploring the usefulness of this variable as a way of identifying addicted cigarette smokers.

The category of nonsmoker is also in need of refinement (*49*). Little attention has been given to developing a systematic typology for nonsmokers, although self-reported reasons for not smoking have been compiled. A typology of nonsmokers may prove useful and may help guide researchers to particular subsamples of nonsmokers in order to evaluate specific hypotheses. For example, some nonsmokers have never even tried a single cigarette and, hence, their own positive or negative biological responses to smoking cannot influence their recruitment to smoking; psychosocial factors in such cases might be said to have precluded the involvement of biological influences on becoming a smoker (*46*). These biologically-uncontaminated "never smokers" are ideal subjects for studies on psychosocial influences on smoking/not smoking.

Multiple Drug Use

One of the most reliable correlates of cigarette smoking is the use of other drugs. Smokers consume more coffee (caffeine), more alcohol, more psychotropic drugs, more marijuana, and more aspirin than do nonsmokers (*1*). The correlations between the various drug uses can be difficult to interpret. Consider the conditional probabilities of drug use in a large sample of U.S. college students in 1969–70 (*33*). If a student used tobacco, the probability was .97 that the student had used alcohol; if alcohol, the probability of tobacco use was .62. If marijuana was used, the probability of tobacco use was .77; if tobacco, the probability of marijuana was .44. With such figures in mind, it becomes foolhardy to ignore possible multiple drug effects when studying any one drug.

The psychosocial pressures for adolescents to use one drug are similar to the pressures to use others (*31*). Kandel (*41*), in a large-sample study of adolescents in New York State, found that peer pressures had consistent and strong effects on drug use (marijuana, tobacco, alcohol, barbiturates, tranquilizers, and stimulants). Significant patterns of intrafamilial multiple drug use have been noted (*3*). Further, in a large longitudinal study (*42*), Kandel found systematic patterns of paths from one drug use to another. For example, though most respondents started with beer or wine, some went on to cigarettes next, while some went on to hard liquor. From either branch, liquor or cigarettes, some individuals went on to marijuana, while some persons became both liquor drinkers and cigarette smokers before trying marijuana. The conclusions of this study have important methodological implications:

> Whereas most studies compare youths within a total population on the basis of their use or non-use of a particular substance, my results suggest a different strategy. Since each style represents a cumulative pattern of drug use and generally contains fewer adolescents than the preceding stage or stages in the sequence, comparisons must be made among members of the restricted group of respondents who have already used the drug or drugs at the preceding stages, and those who have not. Unless this is done, the attributes identified as apparent characteristics of a particular class of drug users may actually reflect characteristics important for involvement in drugs at the preceding level (p. 914).

Kandel's suggestion demands large-sample research, and the larger the number of drugs of interest (for example, caffeine should probably be added), the larger the samples will have to be.

The methodological significance of the multiple drug use patterns has been clear to epidemiological researchers for years, particularly with respect to smoking (*105*). For example, it has been argued that the apparent association between coffee drinking and heart disease is actually due to an often unmeasured, but nonetheless confounding, correlation between smoking and heart disease (smoking and coffee drinking are positively correlated) (*21*). This interest in the confounding or interactive effects of multiple drug use has been slow to influence behavioral, physiological, or personality studies of cigarette smoking. The methodological implications are clear.

Consider, for example, a laboratory study in which subjects are asked to abstain from cigarettes for an hour before coming to the experiment. Since cigarette smokers are more likely to be coffee drinkers or alcohol drinkers, they are more likely to come to the study with significant doses of caffeine or alcohol in their systems. Without knowing it, the experimenter may be looking at the correlated effects of other drugs on the behaviors of interest. If the researchers deprive all subjects of caffeine well before the start of the study, they would not necessarily solve this problem, but rather they may unwittingly find themselves looking at the differential effects of caffeine withdrawal on their measures (*44, 45*). The effects of confounding drug use even on the filling out of personality inventories are not at all understood.

Social Factors

Family and Peer Pressures

Many of the social factors that are involved in the establishment of smoking are important for the maintenance of the habit. As the young adult begins to leave the direct sphere of influence of the family, presumably the effects of parental and sibling smoking habits (*7, 8, 66, 71*) would weaken; there is no reason to expect, however, that peer pressures to smoke (*66, 71*) will be any less strong during the early years of the individual's career as a smoker. The adult smoker is likely to have many smoking friends (*57*). Probably the most important family structure influence on the maintenance of cigarette smoking derives from the smoking habits of spouses or cohabitants (*53, 95*). A major survey by the American Cancer Society shows that 68 percent of young women smokers have boyfriends or husbands who smoke, compared with only 41 percent of the nonsmokers (*16*). The increasing militancy of nonsmokers and the increasing restriction on public opportunities to smoke (*99*) may be acting to tighten the ranks of cigarette smokers, making the support of a group of smoking friends all the more important to the maintenance of the habit. To our knowledge, no data have been gathered as yet on this point. Brecher and his associates (*10*) have proposed that the illusion that quitting is easy or the illusion that cigarettes are not dependence-producing helps the smoker to maintain the habit in the early years. Indeed, if one believes that cigarettes' damaging effects to health occur only after a long history of smoking and if, at the same time, one believes that he or she will be only a short-term smoker, the health consequences of smoking are, in effect, tabled as a reason for not smoking. Research reported by Green (*32*) isolates what is called a "rationalization factor" which is consistent with the preceding interpretation of what many young smokers believe about their smoking.

Some smokers do feel that there is room for doubt concerning the link between smoking and health. Such beliefs do at least give "rational" support to the maintenance of smoking.

Smokers do seem to gain some benefits from smoking. For example, the smoking typologies, discussed above, which are based on self-reports of why smokers smoke, indicate a range of perceived benefits from smoking. Green (*32*) describes the results of administering tests of the Horn typology to a large sample of smokers in the United States: the Pleasurable Relaxation, Tension Reduction and Craving factors were the most important reasons overall, and the Habit, Stimulation, and Handling factors were of substantial but lesser significance. If smoking can be used to relax or to stimulate the smoker (*63*, *80*), it may genuinely contribute to successful performance in a variety of settings. Mausner (*55*) has discussed some particularly social gains from smoking, arguing that smoking is part of a complex social ritual and that it can be an important expressive behavior which helps to define the individual's self-concept.

Social Class and Social Mobility

In our culture, socioeconomic status, at least as measured by occupation, has had a stable relationship to cigarette smoking (*86*). White-collar workers (professional, technical) have the lowest smoking rates; blue-collar workers (laborers, craftsmen) have the highest smoking rates. Men show this relationship strongly, but women tend to show an opposite relationship. Employed white-collar female workers have a higher incidence of smoking than do the blue-collar female workers.

As Reeder (*68*) has pointed out, two excellent longitudinal studies have shown a relationship between social mobility and smoking behavior. Clausen (*17*) reports that upwardly mobile (relative to parents' SES) men were less likely to smoke; downwardly mobile men were more likely to be heavy smokers. Similarly, Srole and Fischer (*93*) report that for males upward mobility decreases the incidence of smoking, while downward mobility increases the incidence of smoking; the results for females do not show the same pattern and are difficult to interpret.

Sex Roles

One of the most striking findings to have emerged from basic surveys on the incidence of smoking in teenagers is the increase over the past 20 years in smoking among girls. No corresponding increase has been found among teenage boys. The latest survey in this series (1975) shows that teenage girls now equal boys, 20 to 21 percent, respectively, in the incidence of cigarette smoking (*68*). Reeder proposes that correlated changes in the sex role of women, as manifest in changes in

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college attendance and in labor trends, may be responsible. For more discussion of these issues, see the Public Health Service report on cigarette smoking among teenagers and young women (*60*) and the report by Bosse and Rose (*9*).

Cessation of Smoking

Individual Factors

Two basic types of research are relevant to personality influences on stopping smoking. The first type concerns studies which have measured the personality characteristics of those who have become ex-smokers, with no particular regard to how they became ex-smokers. The second type deals with the personality correlates of success in specific smoking treatment programs.

Personality Characteristics of Ex-Smokers

Eysenck's research on British males (*28*) showed that ex-smokers were equal in extraversion to nonsmokers and to light smokers, but lower in this trait than were medium or heavy smokers; neuroticism was unrelated to smoking habits. In a longitudinal study of British men and women, Cherry and Kiernan (*15*) found that low daily cigarette consumption and high extraversion scores were each independently related to a greater incidence of giving up smoking. These relationships held for both men and women. Neuroticism had no relationship to smoking cessation in women, but for men, the more neurotic were less likely to give up smoking. A model was derived which has very impressive predictive powers. For men, neuroticism and extraversion scores were each divided into high and low categories and daily cigarette intake at age 20 was divided into three categories (1-10, 11-20, 21+). It was predicted that 47 percent of the high extraversion-low neuroticism-low consumption individuals would stop smoking, and 50 percent, in fact, did. Only 2 percent of the low extraversion-high neuroticism-high consumption individuals were predicted to give up cigarettes; none did. This study demonstrates the advantage to be gained from considering sex differences and from looking at more than one personality variable at a time.

In a small sample study (N=182) of college undergraduates, the Edwards Personal Preference Schedule (EPPS) showed that former smokers (N=22) expressed aggression more openly than either nonsmokers or smokers who never tried to stop; that they had a stronger need for achievement than any other group, including smokers who had tried to stop but failed; that they had a weaker need for close ties with peers (affiliation); and that they had more behavioral stability than the other groups (*101*). It should be noted, however, that this study failed to replicate EPPS differences that have been found for smokers versus nonsmokers.

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Internal-External Locus of Control

It is not surprising that this dimension has made its way into several studies on this topic. "Internals" should believe in their own willpower and ability, while "Externals" should be much more fatalistic in outlook. One might therefore predict that Internals would be more successful than Externals in the efforts to quit smoking. Straits (*95*) and Foss (*30*) confirmed this prediction; Lichtenstein and Keutzer (*53*) and Burton (*12*) failed to confirm it. A third study showed only complicated interactions between type of treatment technique, Internal-External scores, and success at abstinence (*6*).

Extraversion and Neuroticism

Using general definitions of these two traits, it is possible to see a fairly consistent pattern of results which suggests that neuroticism and, in a more complicated way, extraversion are associated with ability to abstain from smoking. In a longitudinal study of Harvard males, McArthur, et al. (*56*) found slight indications that the heavier smokers who were able to give up cigarettes were best described as sociable and as having strong basic personalities, in other words, high in extraversion and low in neuroticism. Guilford (*34*) found that male quitters were less neurotic than those who were unsuccessful at quitting; this trend was not found in female smokers. In addition, male quitters were more sociable (an extraversion factor); this trend, too, was not found in women. Straits (*95*) found no relationship between extraversion and neuroticism, as measured by Eysenck's scales, and quitting. On the Cattell 16PF questionnaire, male quitters were less tense (that is, low in neuroticism) and had more "critical" and "independent" minds (perhaps this can be seen as more internal locus of control); female quitters had lower "tension" and "apprehension" scores (that is, low neuroticism) (*70*). Jacobs (*39*) found that successfully abstaining males were less "impulsive, defiant and manifestly distressed" and also were less "constricted, guarded and isolated." These two sets of traits were positively correlated with each other ($r(102) = .24, p < .05$); it is not obvious how an "impulsive, defiant" person could at the same time be "constricted" and "guarded." Perhaps the last two components, "manifestly distressed" and "isolated", account for the greatest share of the variance in this association. In a 5-year follow up of a smoking withdrawal clinic (*103*), neuroticism as measured by an emotional status score and by a psychosomatic symptom score was related to quitting smoking; successful abstainers were less neurotic. Ryan (*77*), using the 16PF, found that the upper class male quitters were less neurotic and more extraverted; the lower class males did not show the same pattern, but the sample size of quitters here was very small (N = 11).

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Self-Reported Reasons for Stopping

Four main reasons for quitting were identified by Green (*32*) in an analysis of data that had been gathered along with the large survey of adults carried out by the National Clearinghouse for Smoking and Health in 1975 (*61*). Health concerns, of course, weighed heavily as a reason for stopping. There was a desire to gain mastery of the habit which had been controlling their lives. Some smokers had come to believe that smoking was a messy, filthy, smelly habit and, therefore, aesthetic reasons had become prominent. Some smokers said that they were trying to quit because they felt that their smoking was setting a bad example for others who were under their influence, such as children or friends. Green tried to find out if economic concerns (the cost of cigarettes) were a major reason for stopping, but there was little evidence to support such a claim in this study. Perhaps more substantial increases in cigarette cost would have larger effects on attempts at cessation. Horn (*37*) and Russell (*72*) have argued that economic factors can have a major influence. Certainly among younger smokers the cost of smoking is a reason that is often given for wanting to stop (*78, 79*). Young ex-smokers in grades 7 to 12 gave the following reasons for not smoking, beginning with the most common: (1) no enjoyment of or a dislike of cigarettes, (2) health, (3) the influence of others, e.g., a doctor or a friend, (4) aesthetic or moral objections to smoking, (5) the cost of smoking, and (6) the desire to have athletic abilities unimpaired (this was a more important reason among males than females) (*79*).

Green (*32*) speculates that the increasing social pressures against smoking may be creating some new reasons for not smoking. For example, smokers are being made to feel more and more that their smoking is an unwelcome nuisance to other people, and this may motivate some smokers to try to give up cigarettes.

Horn (*37*) emphasizes four aspects of the perception of the health threats of smoking that may be crucial to the decision to try to stop smoking: (1) becoming aware of the threat, (2) accepting that the threat is important, (3) accepting that the threat is personally relevant, and (4) becoming aware that something can be done about the threat. Eisinger (*23*) has found that, of those reporting an acquaintance whose health has been affected by smoking, 27.1 percent quit smoking; only 9.7 percent of those reporting no such acquaintance quit smoking.

Many smokers come to realize that they are dependent on cigarettes; this realization can lead to low motivation to try to quit smoking (*75*). Mausner (*55*) has studied the reasons that successful and unsuccessful abstainers give for stopping smoking. He concludes that, in general, people decide to stop because of an increased expectation of the benefits derived from stopping, rather than because of the fear of the consequences of continuing to smoke. Most smokers believe that smoking is bad. The people who continue to smoke tend to find not smoking more aversive than the prospect of continuing to smoke; those who stop tend to be able to convince themselves that not smoking would be worth the effort (*55*).

Multiple Drug Use

Unsuccessful abstainers from cigarettes, relative to quitters, are likely to be heavier users of other drugs, especially alcohol and caffeine (*34*, *56*, *96*). Little attention has been given to the special problems of people trying to abstain from more than one drug at once or to the possibilities of a user substituting for the absence of one drug by increasing the consumption of another (*45*). Thomas (*96*) analyzed correlates of quitting in light (less than 20 cigarettes per day) and heavy smokers (20 or more per day), and proposed that the greater alcohol and coffee consumption of the heavy smokers—along with higher anger and anxiety scores—made smoking cessation a more difficult feat for them to accomplish. There are some indications of sex differences in the relationship between alcohol intake and successful smoking cessation: among males, heavier drinkers were less likely to quit (*34*, *93*); among females, heavier drinkers were more likely to quit (*93*), or no significant relationship between drinking and smoking cessation was found (*34*).

Social Factors

Social Class

The data on the effects of social class or socioeconomic status on quitting smoking are full of conflict. Eisinger (*23*) in a large sample study found no relationship between education level and smoking cessation. Ryan (*77*) found that among nonstudent males under age 60 (N = 206) in Greenfield, Iowa, successful abstention was much more common in those scored as being in the upper class. In the Midtown Manhattan study (*93*), for men, socioeconomic status was unrelated to becoming an ex-smoker; for women, there was some indication that lower class smokers were less likely to quit (no statistical tests are reported for this), but the authors assert that the sexes are "quite similar on all three SES levels in their smoking to non-smoking conversion percentages." Meyer, et al. (*59*) conclude from a study of approximately 200 individuals in the New York City area that blue-collar workers had less difficulty in quitting than did white-collar workers. An interesting theory was proposed to account for this finding: a member of the blue-collar group was felt to experience less pressure against becoming a smoker than was a white-collar group member; hence, white-collar workers constitute a specially selected group of high-need smokers for whom smoking, from the start, was important enough to maintain in spite of greater interpersonal pressures not to smoke. Unfortunately, this theory may be trying to account for a phenomenon (white-collar smokers have a harder time quitting) that is far from reliable, as witnessed by the preceding review.

Family and Peer Pressures

The weight of evidence indicates that a smoker who has a spouse who smokes will be less likely to be a successful abstainer (*59*, *88*, *95*, *103*). West, et al. (*103*) found that the smoking habits of the smoker's friends, work associates, siblings, mother or father were unrelated to being able to quit. Schwartz and Dubitzky (*88*) indicate that smoking friends can make a smoker less likely to be able to quit. Caplan, et al. (*13*) have described individual differences in a smoker's dependence on social support, not specifically related to smoking; smokers with low work loads and low social support were much more likely to be able to quit than were those with high work loads or with high social support. Smokers with Type A personality (hard-driving, persistent, competitive, involved in work, overloaded with work) were more likely to be unable to quit than those with Type B personality (having opposite characteristics to the Type A). This report is recommended highly for the appropriateness of its use of multivariate techniques to deal with complicated confounding influences on abstention. Eisinger (*24*) found that the "number of former smokers among their 20 best known friends" was directly related to successful abstention.

Sex Roles

Successful abstainers are more likely to be males than females; Eisinger reports 70.4 versus 29.6 percent (*24*). The smaller percentage of females who are able to quit smoking is one of the most reliable findings in the literature (*23*, *24*, *34*, *103*). Bosse and Rose (*9*), using a national probability sample (N = 5,704), tested the hypothesis that the growing convergence of male and female sex roles would lead to a decrease in the difference in male and female rates of smoking cessation. They found that younger male and female smokers were showing equivalent abstention rates; they described this effect as "the equalitarian shift." They found, then, that both age and sex were related to successful quitting, and, in addition, that "knowing someone whose health had been affected by smoking and who had quit" had an even greater effect on quitting.

Profiles of Successful Abstainers

In a cluster analysis performed on 252 male subjects attending a treatment clinic, Schwartz and Dubitzky (*88*) isolated 5 important factors (clusters) that combined to yield 12 types of subject. The first cluster concerned personal adjustment in work, achievement, sex, and social situations. The second cluster combined chronic illness and anxiety along with recent respiratory ailments and use of psychiatric care. Cluster 3 was labeled perception of smoking; low scores here indicated belief in the health dangers of smoking. The fourth cluster was an equivalent to the chronic, habitual, addictive smoking syndrome described by Tomkins (*97*). The fifth cluster combined the Tomkins concepts of negative and positive affect smoking with positive attitudes toward smoking. For a detailed discussion of the 12 types, consult Schwartz and Dubitzky (*88*). These types were determined without regard to success in smoking withdrawal. When success in withdrawal is considered, the types can be reduced to more general groups of successful abstainers. Four of the types contained 60 percent of the continuing successes and only 20 percent of the failures. All these types had good adjustment, low chronic illness and anxiety, and low chronic, habitual, addictive smoking scores. Three of the types contained a significantly lower incidence of treatment successes. These types were distinguished either by very high chronic illness and anxiety or were high in chronic, habitual, addictive smoking. This latter finding underscores the need for more research on the dependence processes associated with cigarette smoking.

Two other factors were shown to discriminate successful individuals from recidivists. Those subjects who had friends or a wife who smoked were less likely to succeed, and those who had lower socioeconomic status were less likely to abstain. Based on earlier sections of this review, the first factor is more likely to be a significant influence on abstention than is the second.

Straits' (*95*) discriminant function analysis generally confirms the pattern found by Schwartz and Dubitzky. The roles of personal adjustment and chronic illness and anxiety in smoking cessation are generally supported by the earlier sections of the present review.

One final point needs to be made. There is mounting evidence, especially in some large sample studies like that of West and associates (*103*), that measures of cigarette dependence (for example, number of cigarettes smoked per day) are directly and often markedly related to increased inability to quit smoking (*15*, *23*, *39*, *89*, *103*).

Some General Psychosocial Influences On Smoking

Mass Media and Smoking

There is little persuasive empirical research available on the effects of television advertising, or its ban, on cigarette sales or on recruitment to the ranks of smoking. Bans on television advertising for cigarettes in several countries, including the United Kingdom, Denmark, Ireland, New Zealand, and Italy, seem to have had almost no effect on per capita cigarette consumption (*52*). A highly technical, econometric analysis has estimated that the 1965 ban on television advertising in the United Kingdom produced a statistically insignificant fall of 3 percent in cigarette consumption (*67*). In Communist countries, smoking is prevalent without advertising of any sort to support it. Four years after the 1970 ban on television advertising in the United States, there was little indication that this mass medium had a major influence on cigarette consumption (*104*). An econometric analysis by Warner (*100*) in 1977 suggested, however, that the sustained antismoking activities, including mass media, that have been conducted since 1964 may have prevented consumption of tobacco from rising even further than it already has.

Whiteside (*104*) has presented an interesting, though speculative, analysis of media influences on smoking. From 1922 to 1952 in the United States, cigarette sales increased 639 percent; over the same period, the population grew only 54 percent. Cigarette advertising, he argues, had a large effect on building the cigarette market. More recently, however, the cigarette market has been in a relatively mature, stable state and has had a much lower rate of growth. As the cigarette industry has asserted, the major action of cigarette advertising now seems to be to shift brand preferences, to alter market shares for a particular brand. Whiteside notes that, when television advertising was banned, the cigarette industry increased its use of direct marketing techniques, such as displays and promotions at the point of sale. This rechannelling of advertising makes it difficult to evaluate the independent effect of the television ban on cigarette sales.

Foote (*29*) proposes that the downturn in per capita cigarette sales in the United States from mid-1967 to 1970 was the result of the increase in antismoking ads on television. The Federal Communications Commission applied its so-called Fairness Doctrine to cigarette commercials in 1967, thereby requiring broadcasters to provide free time for the presentation of antismoking advertising. The application of the Fairness Doctrine led in 1970 to about $60 million of free television air time being provided to antismoking campaigns. After the ban on cigarette advertising, a major source of subsidy was removed from antismoking campaigns and they became a much less common sight on television. Per capita cigarette consumption began to increase again. The correlation between cigarette consumption trends and antismoking campaigns on television is provocative, but Foote's interpretation of this relationship is open to debate.

Economic Pressures and Smoking

Russell (*72*), in a regression analysis study of the relationship between cigarette costs and cigarette consumption, concluded that the smoking by British males was very sensitive to price changes. Such analyses are necessarily complex and, depending on the particular years considered, the correlations between cigarette consumption and cost ranged from -.52 to -.92. Another econometric analysis has challenged Russell's conclusions and suggests that males are relatively unresponsive to price changes and that females are relatively responsive to them (*4*). Discussing both of the above projects and presenting a new analysis of British data, Peto (*67*) concluded that male cigarette consumption between 1951 and 1970 did show marked responsiveness to price changes. Schachter (*81*) has also argued that cigarette cost can have an influence on the composition of the ranks of smokers.

Economists have developed the concept of "elasticity" to refer to the demand for a product as a function of price. The elasticity of product demand is the percent change in consumption that results from a 1 percent price change. Russell's elasticity estimates for cigarettes indicate that for every 1 percent rise in price estimates, consumption fell by .6 percent. According to usual standards, this shows that cigarette demand is relatively inelastic.

Cross-cultural Perspectives

Damon (*20*) has studied the use of tobacco in seven preliterate or primitive societies, four in the Solomon Islands, Melanesia, and three in sub-Saharan Africa. All seven of the societies had access to locally grown tobacco, as well as cured tobacco. Damon was especially interested in evaluating social reasons for smoking. He found that, unless forbidden by religion, all adults smoked as much as possible. Four of the Melanesian tribes and one African tribe did not "report or recognize social factors as a major stimulus or support for smoking." Their dominant motive was personal gratification. Damon argues that physiological satisfaction is the major controlling influence on smoking in these five groups, even though each is aware that smoking is bad for health. The primacy of physiological factors is further supported by (1) the rapid adoption of smoking once it is introduced, (2) its widespread use unless forbidden by religion, and (3) the frequent inability of smokers to go without tobacco for even a few days. Two African tribes did recognize some social uses of tobacco, in addition to the underlying motive of physiological satisfaction. One of these groups, the Bushmen, had incorporated tobacco-smoking into some of their important social rituals. Damon concludes: "On the whole, among these seven societies personal gratification is much stronger than social influence in maintaining the smoking habit."

Personal gratification is often not considered a socially acceptable motive for drug use in the United States (*10*) and probably in many other Western industrialized cultures. The so-called Protestant work ethic is harsh toward such hedonistic motives and is likely to be much milder toward social motives. Perhaps we in industrialized cultures may have cultural "blinders" to the physiological pleasures of smoking and a special cultural need to emphasize social uses of smoking, although recent scientific research on smoking has been moving away from the long-defended notion that cigarettes produce only a psychological dependence and toward the idea that they produce a physiological dependence (*75*, *82*). Conversely, perhaps some of the primitive groups have been biased against recognizing the social uses of tobacco and culturally predisposed to acknowledge the physiological pleasures of smoking.

Recommendations for Future Research

Specific recommendations about future research were made at a few points in this selective review of the literature, but several general points which echo the advice of other authorities (*19*, *22*, *49*, *68*) should be stated. There are multiple psychosocial influences on cigarette smoking. Multivariate research is needed—with as many as possible of the known factors measured within any one project. Only multivariate research can begin to deal with the problems of substantial intercorrelations and interactions among predictor variables. Large samples are needed for reliable multivariate work. Life-span longitudinal projects are much more valuable than one-shot cross-sectional studies. The small amount of longitudinal data already gathered has given us our most unambiguous and interesting information about psychosocial influences on smoking.

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Modification of Smoking Behavior

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Introduction

Since the health consequences of smoking became more evident in the early 1960's, the development of techniques to aid smokers to quit have proliferated. The methods have ranged widely from gimmicks and over-the-counter cessation aids to formal programs and clinics (*368, 376*). Thus, the concerned professional or layman with an interest in assisting smokers in the process of cessation may find it very difficult to decide which intervention strategy is best or most useful. The social relevance of the topic has focused much of the effort in the field toward clinical presentations of what logically appeared to be the best withdrawal techniques or strategies rather than toward careful research to define what strategy, method, or program is most effective in producing long-term successes or positive changes in smoking behavior. Remarkably, a wide variety of interventions has been offered and recommended to the public, but outcome data needed for critical appraisal of them are scarce.

The task of evaluating the relative efficacy of programs and techniques has been very adequately done in numerous past and recent reviews (*24, 26, 29, 40, 171, 200, 224, 226, 230, 245, 366, 368, 376, 413*). Therefore, this review can be selective in order to allow discussion of critical topics and encourage new developments in the field. The reader is referred to the other available reviews to obtain a more detailed discussion of topics that are here given brief treatment.

Methodological Issues

Any reviewer of the literature on strategies to modify smoking behavior is faced with the difficult task of sorting through outcome research that is permeated by many methodological flaws and deficiencies (*24, 26, 224, 226, 366, 368, 376*). Despite the facts that smoking behavior offers an objectively measurable target behavior, that potential treatment participants are numerous, and that the normal treatment context affords the opportunity for both good internal and external validity (*24, 200, 226, 393*), a number of methodological inadequacies continues to plague the field (*26, 29, 226, 368, 376, 413*). Therefore, the methodology and design problems that most commonly limit the appraisal of existing outcome data will be briefly summarized. Anyone concerned with smoking withdrawal programs or research, however, should refer to other comprehensive evaulations of these issues presented by Bernstein (*24*), Schwartz (*366, 376*), Lichtenstein and Danaher (*226*), and the National Interagency Council on Smoking and Health's (NICSH) *Guidelines for Research on the Effectiveness of Smoking Cessation Programs* (*272*).

The most pervasive problem in the evaluation of outcome data from smoking cessation programs is the validity of the treatment results. Almost all clinics and research studies have relied primarily upon unverified self-reports of smoking as their critical dependent measure. Unfortunately, the verbal or written requests for estimates of number of cigarettes currently smoked per unit of time depend upon the participant's accuracy and honesty (*226*), are subject to nonspecific demand characteristics (especially during and after treatment) (*226*), and appear to be highly influenced by digit-bias (that is, given in multiples of 5 or 1/2 pack units) (*423*). One study collecting global estimates under different conditions on the same day found questionable reliability (*423*). Thus, studies based only on global, unverified self-reports of smoking behavior must be viewed with skepticism.

Because of these factors, the rate measure based on such global estimates tends to be more an ordinal than a ratio variable (*396*). Nevertheless, rate-per-unit-of-time data often have been preferred over the dichotomous abstinent-nonabstinent or percent-reduction categories, which clearly require the use of less powerful nonparametric statistical analyses (*226, 393, 396*). The use of self-monitoring recording has been recommended in various forms (*109, 198, 226, 250, 272*) and commonly used in many studies to enhance both the reliability and psychometric qualities of the rate data. However, the procedure is known to be reactive (*198, 250*), is still susceptible to the demand characteristics (*198, 226*), and tends to underestimate the "real" baseline or follow-up rate (*109, 198, 226, 250*).

Studies not relying on smoking rates as the primary dependent measure have commonly utilized various and often undefined success-failure categories to minimize the problems of self-report data (*24, 366*). Standard categories have been suggested to avoid ambiguity (*272*); however, the primary evaluation of treatment-results based on abstinence data can be recommended for several reasons. First, abstinence is the primary goal of almost all smokers seeking treatment (*24, 25, 40, 171, 226, 366*). Second, follow-up data on smokers have indicated that most smokers who fail to attain abstinence eventually return to baseline smoking rates (*24, 26, 171, 251*). Third, analyses of rate data can yield statistically significant treatment effects even with a clinically insignificant proportion of participants abstinent at follow-up (*251, 366, 376*). Fourth, abstinence reports are less susceptible to nonspecific demand characteristics and the reactivity of self-monitoring (*226*). Nevertheless, when derived from reliably collected self-monitoring data, cigarettes-per-day rate data or the more precise percentage-or-baseline (current smoking ÷ pretreatment smoking rate x 100) variable (*199, 200, 226*) can be very helpful as secondary measures for testing finer theoretical questions with parametric statistical techniques (*24, 200, 226, 272*). Because treatment will often produce a marked, positive skewness in the distributions of rates (that is, greatly increased frequency of rates at or near zero), care should be taken to test the homogeneity of variance and to apply transformations as necessary before utilizing analysis-of-variance procedures, especially with cell frequencies of unequal size (*71, 292, 445*).

Optimally, self-report data on smoking should be validated by an objective measure. False reporting has now been documented in both children (*99, 154, 262*) and adults in cessation programs (*47, 82, 178, 283*). Natural-environment informants or observers have been recommended and used in many studies, but the systems are reactive, difficult to maintain, and, owing to possible collusion, have questionable validity (*47, 226*). Biochemical tests for objectively measuring smoking exposure are clearly more desirable. Measurements of blood carboxyhemoglobin (COHb) (*61, 192, 320, 330, 397, 427*) and thiocyanates (SCN$^-$) in biologic fluids (*18, 54, 75, 83, 238, 299, 300, 444*) have been demonstrated to be reliable indicators of smoking behavior. Concentrations of carbon monoxide (CO) in alveolar air is directly proportional to blood COHb concentrations (*61, 320, 330, 397*) and has been recommended as a simple validating tool (*208*). However, CO concentrations have a very short half-life (*330, 397*) and show high diurnal variability (*61, 258, 330*). Thus, SCN$^-$ concentrations that have a biologic half-life of approximately 14 days (*299*) are more suited for validation of self-reports (*47, 54, 423, 424*). Determinations of serum SCN$^-$ have been more common (*47, 54, 83, 423*), but tests of urine or saliva are also possible and may be more practical in many clinical settings (*18, 99, 262*). Unfortunately, COHb levels are affected by various environmental exposures (*192, 397, 427*) and SCN$^-$ concentrations can be elevated by diet (*47*). Singly, however, they provide a crude measure of smoking rate (*423, 424*) with adequate discrimination between smokers and nonsmokers; together they appear to provide a very powerful test of abstinence (*423, 424*).

In summary, researchers should be aware that uncorroborated self-reports may lead to an overestimation of success, especially in situations where subjects are under social pressure to quit or to report quitting. The addition of objective biological assays can help to validate self-report data and improve the ability to assess outcome, using the self report as a low-cost, easily obtainable, dependent measure.

In addition to the problem of questionable validity of self-reports that faces all researchers, various design deficiencies also plague the field (*24, 200, 226, 272, 304, 366, 367, 376, 398*). First, attributions of causality of outcome results to independent treatment factors are virtually impossible without systematic designs, including appropriate experimental controls (*24, 56, 391*). Initial demonstrations of efficacy may be evaluated relative to commonly expected norms of success (*245, 304*); such clinical demonstrations must then be replicated versus appropriate control conditions, especially attention-placebo controls (*24, 26, 200, 226, 230, 245, 251, 272, 304, 366, 367, 376, 398*). Few procedures or programs developed in clinical settings have progressed to experimental validation (*24, 40, 245, 304, 366, 367, 376, 398, 413*). Moreover, Straits (*398*) has suggested that the strength of laboratory research involves testing more complicated questions than treatment efficacy. Factorial designs enable one to evaluate specific treatment effects as well as more complex multidimensional and interactional effects and thus permit the simultaneous testing of several theoretical issues (*398*).

Systematic treatment evaluations must also include comprehensive and adequate follow-up of participants (*24, 26, 171, 272, 366, 368, 376*). Almost all treatments are able to show dramatic post-treatment effects, but rapid relapse in most participants has been the norm (*170, 171, 251, 366*). Therefore, no treatment can be adequately evaluated without long-term follow-up data. Recidivism tends to be the greatest during the first 3 to 4 months after treatment and relatively slight after 6 months (*170, 171*), but a 1-year follow-up remains highly recommended (*272, 366, 368, 376*).

Comprehensiveness of follow-up is as important as length, if not more so. Schwartz (*366, 368, 376*) has strongly emphasized that all participants, including early-treatment dropouts, should be used in computing treatment effectiveness. Additional analyses of subjects completing most treatments are useful to clarify theoretical issues (*24, 226*); however, the relative efficacy of the procedure should be judged on the stricter standard (*272, 366, 368, 376*). Follow-up results based only on participants who respond or who are readily available are especially suspect (*24, 272, 366, 368, 376*).

The final issue that commonly affects outcome data from smoking-modification studies involves the replicability and generalization of results. Programs and studies with reportedly very similar procedures have produced highly variable patterns of results (*24, 26, 40, 171, 200, 226, 230, 366, 376, 413*). This, it seems, is due in part to the variability introduced by small samples and population differences (*24, 171, 226, 272*) and the inadequacies of theoretical models guiding the descriptions of treatment variables (*24, 272, 306, 398*). In an effort to minimize these deficiencies, the NICSH *Guidelines* (*272*) stress the need to describe completely the recruitment and selection of participants, their characteristics, and the specifics of each aspect of treatment. Keutzer, et al. (*200*) have also discussed the problems of uncontrolled variability from group treatment and inexperience of the therapist or experimenter.

Thus, conclusions regarding the relative efficacy of treatments can be reliably made only when methodological deficiencies are at a minimum (*272*). The quality of the data has improved markedly since the early reviews (*24, 200, 366*), but almost all studies remain deficient in some respect (*368, 376*). Many programs have collected little or no objective follow-up data, and the lack of methodological rigor compromises the results of many others that have. Therefore, based upon current data, the replicability and general utility of almost all procedures can be only tentatively assessed.

Review of General, Nonspecific Interventions

A variety of interventions has been developed and offered with the primary goal of aiding a group of smokers to become nonsmokers rather than testing how the procedures may work (*398*). Various reviewers have analyzed the data on this type of intervention, which includes public service and proprietary withdrawal clinics, individual or medical counseling, and large scale coronary prevention trials. Except for the coronary prevention trials, the clinical-treatment focus of these interventions has resulted in multiple uncontrolled clinical replications, often without adequate outcome data (*24*, *40*, *171*, *200*, *245*, *366*, *368*, *376*). Additionally, the vast public health campaign of recent years should be considered as a special class of general, nonspecific interventions both to prevent smoking onset and to stimulate cessation (*24*, *40*, *200*).

Public Health Educational Campaigns

The public health campaign against cigarettes has produced notable changes in public awareness of the health consequences of cigarette smoking (*175*, *269*, *271*, *422*). It appears that the dramatic changes noted in adult smoking, especially among middle-aged males and certain professional groups (*86*, *100*, *121*, *271*, *421*), can be attributed largely to the effectiveness of information and educational campaigns since 1964 (*130*, *270*). Moreover, Warner (*428*) has estimated that the effect of specific "events," such as the 1964 Surgeon General's Report, on cigarette consumption (mean number of cigarettes consumed per day) may appear small and transitory, but that the cumulative effect of persistent publicity appears to have reduced consumption by 20 to 30 percent below its predicted 1975 level.

More specifically, O'Keefe (*284*), in a study on the impact of television anti-smoking commercials during the late 1960's, revealed changes in attitudes and reported reductions in consumption but little direct impact on smoking cessation. Forty-two percent of those motivated to quit felt the commercials acted as an incentive, but only 1 percent of the ex-smokers credited the commercials with helping them quit. Similar minor effects were noted in a smaller trial with anti-smoking posters (*5*). Ryan (*353*) reported the results of an entire community's attempt to quit in 1970. Thirty-seven percent of the adults attempted to quit, and 14.2 percent of the males and 3.9 percent of the females were still reporting abstinence 7 months later, with higher socioeconomic groups being more successful. The Avdel smoking project (*98*) also seemed to have produced small but meaningful changes in both smoking attitudes and behavior with a worksite campaign. These specific and general results of the public health campaigns appear very similar to other British (*343*) and worldwide experiences (*130, 301*).

Public Service and Proprietary Clinics

It is interesting to note that Bernstein's (*24*) comment that the educational campaigns have affected research and clinical activities more than smoking behavior still seems valid. Public service and proprietary programs have proliferated since 1964. Schwartz and Rider (*376*) have provided a summary of the published and unpublished data on these types of programs. Many such smoking-withdrawal clinics offered by voluntary agencies have been intermittent and rarely evaluated. The group program of the American Cancer Society (ACS) (*2, 3, 160*) and the 5-Day Plans of the Church of the Seventh Day Adventists (*252, 253, 254*) have, however, remained very active in providing public service treatments to smokers. Unfortunately, while the two programs together have probably helped more smokers than any other organized effort (*245, 368, 376*), only limited published outcome data are available for consideration.

The 5-Day Plan has become standardized and involves five consecutive 1½- to 2-hour sessions focusing on immediate cessation, and dietary, physical, and attitudinal changes to reduce withdrawal effects (*252, 254*). Because of its clinical focus, almost all evaluations have been without controls (*117, 146, 147, 148, 213, 252, 253, 254, 267, 298, 366, 376, 403, 412*), with good immediate abstinence rates of approximately 60 to 80 percent, but with an approximately 50 percent relapse by 1- to 3-months post-treatment. Unfortunately, clinical claims of abstinence among 33 to 40 percent of participants beyond a year post-treatment (*146, 147, 148, 253*) are markedly discrepant from other clinical demonstrations (*213, 267, 298, 361, 412*). Guilford's comparative study of the 5-Day Plan (*137, 138*) found abstinence rates of 16 to 20 percent at 1 year that may not differ from unaided attempts (*137, 138, 412*). Nevertheless, the program appeared to be more successful with males (*137, 138, 267, 403*) and when higher expectation of success was reported by participants (*361*). Results of all studies are based on unverified self-reports, often only from subjects completing all treatments (*366, 376*).

Available long-term abstinence outcome data on the ACS group programs (*2, 3*) also appear to be somewhat disappointing. The one available evaluation of the ACS groups, which focus on insight development, group support, and self-selected cessation techniques, was conducted on 29 clinics in Los Angeles from 1970 to 1973 (*318*). Telephone follow-ups were completed on 354 subjects selected from a random sample of 487 of the original 944 participants. Abstinence rates based on the total random sample were 41.7 percent at post-treatment, and 30 percent at 6-month, 22 percent at 12-month, and 18 percent at

18-month follow-up points (*245*, *318*, *378*). In the subsample group of 354 subjects who were contacted (*318*), 28.4 percent of the males and 20.3 percent of the females reported abstinence.

Other clinics with similar or more elaborate formats have reported fairly equivalent outcome data (*63*, *81*, *82*, *114*, *158*, *178*, *213*, *274*, *286*, *289*, *433*, *438*, *440*, *448*). The Smoking Withdrawal Study Centre in Toronto (*81*, *82*, *378*) used comprehensive educational groups with 472 smokers and obtained successful abstinence in 28.6 percent of all participants at 1-year follow-up, with 33.9 percent of the men and 20.8 percent of the women being successful. However, carboxyhemoglobin (COHb) assessments revealed that 22 of the 107 (20.6 percent) reported ex-smokers had levels over 5 percent, which strongly suggested smoking. A 5 percent quit rate was noted among a no-treatment control group. In a population based sample, Isacsson and Janzon (*178*) were able to produce abstinence during an intensive 6-week program among 31 of 51 participants (60 percent), with 17 (33 percent) remaining nonsmokers at 8- to 9-month follow-up. Abstinence was verified by COHb determinations. West and his colleagues (*433*) followed up 559 smoking-cessation clinic participants 5 years later and found 17.8 percent of the contacted sample reporting abstinence. Approximately two-thirds of those who had quit during the clinic had returned to smoking, while only 8 percent of the unsuccessful participants were reporting abstinence at follow-up. Older males who had lighter smoking habits and more stable environments appeared to be most successful. Research clinics (to be discussed in more detail elsewhere in this report), offering similar treatment formats, have reported similar 15 to 20 percent long-term abstinence among participants (*341*, *373*, *374*, *380*, *381*, *382*).

In light of these data on public service and research withdrawal groups and clinics, the claims of more impressive results by proprietary programs must be viewed with caution (*116*, *245*). Schwartz and Rider (*376*) reviewed a variety of unpublished data on commercial methods, but only one published evaluation of a commercial method is currently available. In this study (*194*), records of 553 participants of the SmokEnders program in 1971 were examined and a $3^1/_2$- to 4-year follow-up was attempted on the 385 (70 percent) who were not smoking at treatment termination. Only 167 (43.4 percent) were contacted; of these, 57 percent of the males and 30 percent of the females were not smoking. Schwartz and Rider (*376*) noted, however, that, even if the smoking rates of those contacted at follow-up accurately represent the total successful sample, the long-term success based on all participants (including treatment dropouts) would be about 27 percent rather than the reported 39 percent. As the men and women were reported to have been about equally successful at treatment termination, the higher follow-up success rate for males would still seem valid.

In viewing the data from many clinics relative to the 16 to 19 percent success at 1-year follow-up noted in Guilford's (*137*, *138*) and Schwartz and Dubitzky's (*373*, *374*) unaided control groups, the impact of many programs appears to have been minimal. Bernstein's (*24*) conclusion still seems valid: clinics can serve a very useful purpose when more effective modification techniques are developed for general distribution, but uncontrolled use of nonvalidated notions cannot refine those procedures. The attempts to analyze more carefully the clinic format has produced some enlightening data (*81*, *82*, *137*, *138*, *178*, *318*, *341*, *361*, *373*, *374*, *380*, *381*, *382*, *433*). Long-term results imply that males in these clinics fare better than females during maintenance (*81*, *82*, *137*, *138*, *267*, *341*, *376*, *403*, *433*). Moreover, the comprehensive follow-up and physiological validating of some studies (*81*, *82*, *178*, *373*, *374*) highlight how misleading early success based on self-reports can be. The placebo effect noted in control groups highlights the fact that many of the treatment effects of clinics remain undefined (*373*, *374*). More effort should be made, therefore, to evaluate on-going clinical activities so that researchable hypotheses can be illuminated for further controlled study (*24*, *394*).

Individual and Medical Counseling

Smoking-cessation counseling by professionals in private practice is known to exist, but published data on its efficacy are very rare. A report on two psychotherapist-led groups suggests that long-term therapy may help some smokers (*39*); however, the cost of such treatment would seem prohibitive (*245*). In controlled studies of the type of individual and group counseling formats that could be easily and less expensively disseminated, Schwartz and Dubitzky (*373*, *374*) and the American Health Foundation (*380*, *381*, *382*) produced 1-year abstinence rates ranging from 13 to 30 percent with no clear superiority for individual or group therapy. While individual counseling styles seemed to affect initial success and dropout rates, there were no differences in effectiveness during follow-up (*186*, *431*).

Since smokers have become almost uniformly aware of the health risks of smoking (*269*, *271*, *422*), they view the physician as an important person in the quit-smoking decision (*271*). However, only about 25 percent of smokers surveyed in a national telephone interview reported having been advised by their physician to quit (*271*). Almost all physicians are convinced of the health consequences of smoking and have made dramatic changes in their own smoking (*121*, *421*), but many seem reluctant to confront their smoking patients until serious effects are present (*55*, *338*). Nevertheless, numerous studies of ex-smokers have shown that linking the increase of symptoms, such as coughing or breathlessness, to smoking was a major precipitant for unaided quitting (*51*, *128*, *150*, *152*, *190*, *294*, *389*, *390*, *399*, *400*, *418*, *419*).

Rose (*338*) and Lichtenstein and Danaher (*227*) have reviewed the issue of physician counseling and its efficacy. In general, it appears that physicians have been discouraged from this role (*338*) and are effective as counselors only when dramatic symptoms are present (*227*, *338*). Several uncontrolled studies, done primarily in England, have shown varying success. Early studies in this country showed minimal effects (*244*, *322*). Studies abroad, on the other hand, have evaluated several important aspects of the process. Porter and McCullough (*312*) produced only 5 percent abstinence at 6 months in a briefly-counseled group, while 4 percent quit in a randomly defined uncounseled group. Handel (*153*) reported more impressive results from one brief session with 17 of 45 (38 percent) males and 6 of 55 (11 percent) females reporting abstinence at 1-year follow-up. When patients presented current respiratory symptoms, Williams (*443*) and Burns (*51*) found a higher response to brief counseling. Burns (*51*) reported 35 of 66 (53 percent) males and 9 of 28 (32 percent) females reporting completely stopping 3 months after the visit. Similarly, Williams (*443*) found that, of 204 patients routinely counseled, 59 of the 160 (37 percent) who could be contacted at 6-month follow-up were reporting abstinence, with males and females being about equally receptive.

Some of the variability of response may be due to individual physician styles. Pincherle and Wright (*302*) followed up a total of 1,493 business executive smokers for 1 to 2 years after a regular physical where smoking-cessation advice was given. Thirteen percent reported quitting and 11 percent indicated a reduction in rate of 30 percent or more; however, when the results were analyzed across various physicians giving the message, success (quitting or 30+ percent reduction) rates varied from 35 percent to 17 percent. In a similar follow-up of antismoking advice given during annual physicals, Richmond found 118 of 543 (22 percent) quit for at least 1 year; 15 subsequently relapsed, leaving a long-term success rate of 19 percent (*329*). Unfortunately, no physician-counseling study has utilized techniques to validate self-reported behavior change.

Considering the brief nature of the contact and the lack of specific maintenance follow-up, the reported rates of abstinence seem encouraging. A study by Raw (*319*) has suggested that both a physician's message and counseling by a health professional in a white coat were important in producing cessation, also suggesting that health professionals other than physicians should become more involved. Peabody (*291*) reported that with a well-developed program, 25 percent of smokers will quit after the initial counseling, 25 percent will quit after several attempts, 20 percent will eventually stop with difficulty, and only 30 percent will never respond. These expectations may be high for a general patient population, but cessation data on special groups of patients with current medical problems related to smoking are encouraging.

Patients hospitalized with their first myocardial infarction (MI) provide a dramatic example of this. Thirty to fifty percent of the smokers in this group permanently stop smoking after only routine advice (*4, 11, 68, 157, 338, 430, 432, 442*). Follow-ups on hundreds of such patients reveal that relapses back to smoking are uncommon, with 50 percent quit rates often maintained for 1 or more years (*11, 68, 338, 430, 432*). When more intensive counseling and active follow-up support were undertaken in a study by Burt and associates (*52*), 70 of 114 (61 percent) of cigarette smokers and 9 of 11 (82 percent) of cigar and pipe smokers stopped smoking after hospitalization, and only 19 (15 percent) of the smokers made no changes. At the 1-year follow-up, 9 of the immediate quit group (11 percent) and 13 of 22 (59 percent) who quit later relapsed, leaving 79 of 125 smoking (cigarette, pipe, or cigar) patients reporting abstinence (63.2 percent) with 27 (21.6 percent) having reduced. Among 120 patients given conventional advice and not followed up in the special clinic, only 27 of 98 (27.5 percent) of the smokers were reporting abstinence and 27 (27.5 percent) reporting reduction at the 1-year follow-up.

Thus, physicians and other health professionals have great opportunities for anti-smoking counseling. Both Rose (*338*) and Lichtenstein and Danaher (*227*) warn, however, that the private practitioner should avoid unrealistic expectations and underestimations of the time required. Various guidelines have been offered on the office management of cigarette smoking (*113, 115, 166, 291, 307, 309, 402*); Lichtenstein and Danaher (*227*) provide a comprehensive format and suggestions. Clearly, health care professionals can play a dramatic role by being nonsmoking models, by linking current symptoms to smoking, and by aiding smokers in the decision to quit alone or with additional help. But as Rose (*338*) and Lichtenstein and Danaher (*227*) have pointed out, additional research is needed to test techniques applicable for office-guided cessation programs.

Large-Scale Coronary Prevention Trials

Middle-aged men judged at risk but not exhibiting coronary heart disease (CHD) provide a special challenge for smoking counseling (*336, 337*). Since cigarette smoking together with serum cholesterol and blood pressure levels are considered the major risk factors for CHD (*36, 420*), preventive trials have attempted to reduce the incidence of CHD in study samples by using a multifactor approach. The Coronary Prevention Evaluation Program (*391, 392*) was an initial 7-year feasibility test of this approach among 519 coronary-prone men aged 40 to 59 at intake. Only 116 of the original 191 smokers remained active in the study, and more emphasis was given to nutritional counseling than to smoking counseling. Nevertheless, 43 of the 116 (37.1 percent) remaining smokers eventually stopped smoking.

Subsequently, other trials were initiated in Europe (*449*). Wilhelmsen (*439*) established a comprehensive cessation program for use in a field trial in Sweden (*441*), but long-term results are not available. In a controlled trial of the effects of anti-smoking advice among 1,470 coronary-prone London civil servants (*324*), 51 percent of the 714 randomly assigned to anti-smoking clinics stopped smoking by the end of 1 year. Only 31 percent were reporting complete abstinence, as many converted to pipes and cigars (*338*). In general, the preliminary results of the European multifactor prevention trials are only moderately successful, with abstinence in 16 to 28 percent of the smokers after 1 year (*449*).

In 1972 the Multiple Risk Factor Intervention Trial (MRFIT) was initiated in this country (*265*, *266*). One of the largest and most ambitious of the multicomponent efforts to influence cigarette smoking behavior among middle-aged men, this smoking intervention attempt is occurring within a broad 6-year coronary prevention program also intended to reduce serum cholesterol and blood pressure levels in over 6,000 men aged 35 to 57 at increased risk of coronary disease (*410*). Initial intense intervention involving multicomponent group or individual sessions produced abstinence in approximately 43 percent of the smokers by the first annual examination (*280*). Biochemical assessments are being made to validate the self-report data. Continued intervention and maintenance contacts have produced successful cessation in other participants who had not formerly quit and in participants who had returned to smoking (*280*).

Two studies have focused on total populations rather than selected high-risk groups. The North Karelia Project (*204*, *316*) has been providing a comprehensive community program since 1972 to reduce the very high rate of cardiovascular disease in eastern Finland. By the end of the first year of intervention, the proportion of males aged 25 to 59 in the North Karelia district who smoked decreased from 54 percent to 43 percent, while female smoking rates have remained at about 11 to 13 percent throughout the 5 years of treatment. These encouraging changes in male smoking behavior were maintained, with the 5-year follow-up survey reporting 42 percent of the adult men still smoking.

More specific data are available on the field study conducted by the Stanford Heart Disease Prevention Program. An extensive 2-year, mass-media campaign (*234*) was presented to two California communities to persuade the general public to modify eating and smoking behaviors in order to reduce cardiovascular risk. A third community served as control (*101*, *235*). Face-to-face behavioral counseling (*101*, *247*, *258*) was offered to two-thirds of the high-risk subjects in one of the media communities. Three years after the program started, the proportion of smokers had decreased by 3 percent in the control community, by 8 percent in the media-only community, and by 24 percent in the media-plus-counseling communities (*101*, *248*, *259*). Fifty percent of the high-risk smokers receiving face-to-face counseling, but only 11 percent receiving just media, had quit (*101, 248, 259*). Thiocyanate monitoring was performed to validate self-reports.

When the risks of smoking are made more immediate and salient, and both skills and support to change are provided, meaningful reductions are possible. The multifactor trials reveal that when smokers are sufficiently educated regarding their risks, they respond much like the post-MI patient and quit immediately and relapse less than would be predicted. The most successful multifactor trials have involved expensive face-to-face intervention techniques and extensive follow-up contacts (*280, 410*) or costly and well-conceived behavioral and media programs (*101, 204, 235, 247, 316*). Hence, more work is needed to translate the skills developed from these research trials into office practice and public health campaigns (*227, 338*). It should be noted that the effective programs involved face-to-face intervention techniques which were both intensive and expensive.

Controlled Experimental Research on Intervention Strategies

A wealth of research data relevant to the modification of smoking behavior has been produced. Early controlled research tended to produce unimpressive results (*24, 200, 366*). Schwartz and Dubitzky (*373, 374*) conducted an exemplary study of what appeared to be the best treatment options available in the late 1960's (*24, 200, 366*). Initial results suggested that group or individual therapy had moderate effects on smoking; but, by the end of a 1-year follow-up, not one of the seven experimental conditions was superior to the no-contact or minimal-contact controls (*373, 374*). Recent progress has begun to highlight both what strategies may be more effective and why they may work. Because these data have been comprehensively evaluated and discussed in recent reviews (*26, 29, 226, 245, 368, 376*), this section will emphasize primarily the major trends in this research history.

Drug Treatments

The psychopharmacology of smoking and its relationship to smoking behavior and cessation are discussed in some length elsewhere in this report and in recent reviews (*46, 136, 181, 183, 349*). While research (*349, 359, 360*) continues to suggest that there are pharmacological determinants for smoking, the identification of chemical agents either to substitute for smoking or to minimize withdrawal symptoms has been frustrating and difficult (*136, 181, 183*).

Early research on Lobeline as a nicotine substitute was equivocal (*24, 200, 366*). The utilization of the substitute in a clinic format seemed to at least enhance short-term effectiveness (*93, 341*), but the double-blind study by Davison and Rosen (*77*) indicated that Lobeline was no more effective than an appropriate placebo. More recently, a nicotine chewing gum has been developed and tested as a cessation aid (*41*, *102*, *103*). Double-blind studies using the gum in cessation clinics suggested that it is significantly more effective than placebos (*41*, *185*, *283*, *352*), but, beyond the control of withdrawal symptoms (*364*), its effects appeared to be a small component in the overall success (*352*).

Combinations of drugs to reduce withdrawal symptoms have been used in various clinics (*180*, *341*, *438*, *440*); however, the double-blind study by Schwartz and Dubitzky (*373*, *374*) of meprobamate with and without individual or group therapy suggested that the placebo, if anything, was more effective. While all treatment conditions were initially superior to questionnaire and screened no-treatment controls, the prescription-only and prescription-plus-individual-counseling had lower (8.3 percent and 13.9 percent) abstinence rates at 1-year follow-up than the controls (16.7 and 19.4 percent) (*373*, *374*).

Other chemicals have been tested in Europe with some initial success (*136*, *363*), but additional evaluations are needed (*136*, *376*). Rosenberg (*340*) reported initial success in reducing consumption in a double-blind study of an antismoking chewing gum that caused an unpleasant taste when tobacco was subsequently smoked. The gum's efficacy as a cessation aid was not tested. Current data suggest that the usefulness of pharmacological cessation aids has yet to be unequivocally demonstrated. While aids such as nicotine gum may be useful in the control of withdrawal symptoms in some smokers, current research suggests that they would need to be combined within a broader program to produce and maintain abstinence (*136*, *352*).

Hypnosis

Clinicians have claimed from 42 to 86 percent of their clients treated with hypnotherapy were abstinent at 6- to 12-month follow-up (*66*, *67*, *143*, *278*, *358*, *395*, *429*, *450*). Unfortunately, these claims have not been substantiated in controlled research. The early research was chaotic and methodologically poor, leading Johnston and Donoghue (*189*) to conclude that "there is almost no good research evidence attesting to the effectiveness of hypnosis in the elimination of smoking behavior" (p. 265). Moreover, Spiegel, a leading proponent of self-hypnosis, claimed that the actual success rate may be closer to 20 percent long-term abstinence (*387*, *388*). Orne (*285*) considered both the theoretical foundations and research data for hypnosis and concluded that its effects can best be categorized as a placebo response which leads to nontraumatic cessation through both the mystique of the procedure and the hypnotic suggestions.

The data from several recent studies do not refute these conclusions. Pederson and associates (*295*) found that 9 out of 16 (54.3 percent) of the subjects in a hypnosis-plus-counseling group were reporting abstinence at 10-month follow-up as compared to 12.5 percent for counseling-only or waiting-list control groups. As there was only 8 percent abstinence for a group treated with hypnosis only, they concluded that hypnosis can enhance the effects of group counseling; alone, it may be insufficient as a cessation procedure. When Shewchuk and associates (*382*) allowed smokers attending clinics to choose group therapy, individual therapy, or hypnosis, 193 of 571 (34 percent) chose hypnosis. The group therapy-reported abstinence rate (49 percent) was significantly superior to those of both hypnosis (38 percent) and individual counseling (33 percent) at treatment termination. By 1-year follow-up, however, all three conditions showed marked relapse, leaving only 17 to 21 percent of the participants reporting abstinence. While assignment to conditions was self-selected and nonrandom, the failure of hypnosis to replicate clinical claims remains important.

Barkley and associates (*18*) found that group hypnosis did not significantly differ from an attention-placebo control in mean smoking rates at any point during treatment or follow-up, but it had more subjects claiming abstinence at the 12-week follow-up point (4 of 8 vs. 1 of 9). At the 9-month follow-up, only two of eight (25 percent) of the hypnosis subjects were reporting abstinence versus none for the control. Francisco's (*105*) unpublished dissertation appeared to have reached a similar conclusion. It has been suggested that a 15 to 20 percent success rate for hypnosis may reflect the expected proportion of subjects highly susceptible to hypnosis (*297*).

Social Psychological Approaches

Higbee (*159*), Leventhal (*216, 217, 218, 219*), and Rogers (*332*) have reviewed most of the data from field and laboratory studies conducted to test responsiveness to persuasive communication regarding cigarette smoking. While most studies on smoking have produced attitude changes without marked or lasting reductions in smoking behavior (*181, 182, 231, 239, 244, 303, 321, 401*), this area of research has clarified several basic aspects of the smoking cessation process. The results and implications of these studies have been summarized by Leventhal (*216, 217, 218, 219*) and Rogers (*332*).

Janis and Hoffman (*181*) demonstrated the facilitating effects of daily telephone contacts that persisted well into follow-up despite termination of the contacts. Unfortunately, mean-rate reductions rather than abstinence rates were reported. Rogers and associates (*333, 334*) have recently documented the long-term impact of several communication strategies on smoking behavior. They reported significantly higher abstinence for high-fear versus low-fear messages in a college sample at 3-month follow-up (22 percent vs. 7 percent), and in a community sample at 1-year follow-up (18.8 percent vs. 0 percent).

Suedfeld's unexpected results with a single exposure to 24-hour sensory deprivation (SD) are also impressive (*405, 406, 407*). In a pilot study with five subjects, four quit after treatment and were reporting abstinence for 1 to 3 months afterwards (*406*). In a controlled study

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(*407*), almost all SD subjects were reported to be abstinent at treatment termination, and 10 of 37 (27 percent) appeared to remain so at 12-month follow-ups when only 4 of 35 (11.4 percent) of control-condition subjects were reporting abstinence. Recently, Suedfeld and Best (*405*) piloted a combination of SD with a complex behavioral program involving aversive smoking and reported abstinence in four of five subjects for over 8 months.

This latter finding is supportive of Leventhal's (*216*, *219*) conclusion that attitude change without a meaningful plan for action will not produce behavioral change. Hence, additional integrations of attitude and behavior change procedures seem worthy of investigation.

Social Learning and Behavior Modification Approaches

Research based on experimental and social learning theories (*12*, *14*, *106*, *168*, *169*, *172*) has produced a wide diversity of controlled studies. Unfortunately, most of the early research on techniques that had been successful with other behavioral problems (*106*) or were derived from the principles of experimental psychology and laboratory research on behavior change proved to be minimally effective in producing long-term changes in smoking behavior. While early reviewers (*24*, *200*, *230*) acknowledged these discouraging initial treatment results, they concluded that the more empirical approach of these procedures made them the most promising. These hopes have been only partially fulfilled (*243*, *451*).

Specifically, many studies have been more concerned with theoretical comparisons based upon evaluations of smoking-rate changes than with developing techniques with documented efficacy based on long-term abstinence data. Techniques were often found to be at least temporarily superior to control conditions, but the effects either vanished during follow-up or no meaningful follow-up was conducted (*25*, *53*, *59*, *64*, *70*, *107*, *132*, *135*, *139*, *155*, *197*, *199*, *201*, *206*, *207*, *209*, *212*, *215*, *220*, *221*, *242*, *255*, *260*, *273*, *276*, *280*, *281*, *287*, *317*, *377*, *384*, *394*, *408*, *409*, *426*, *434*, *435*, *436*, *437*, *447*).

This pattern has been especially common in dissertation research on smoking. Most such dissertation research has been conducted by doctoral candidates and supervised by committees who generally have solid experimental and methodological backgrounds but limited clinical experience with smokers (*225*). Armchair and theoretical analyses of smoking have too often led to experimental and control conditions of some theoretical interest but which typically produced no relative differences among groups at follow-up and weak absolute results as measured by abstinence rates (*225*, *376*). Furthermore, graduation pressures usually lead to insufficient follow-ups of only 1 to 3 months (*225*). The number of unpublished doctoral dissertations of this type document how much well-meaning effort has been devoted to the production of largely inconclusive results (*10*, *20*, *34*, *35*, *38*, *60*, *69*, *87*, *88, 96, 118, 123, 125, 127, 134, 146, 161, 187, 188, 191, 196, 236, 249, 268, 277, 292, 315, 328, 342, 357, 365, 385, 386, 411*).

Overall, the methodology of the research based on learning-theory approaches has been improving (*26, 226, 376*). Most studies have utilized appropriate designs and controls, follow-ups are becoming longer, and, most encouraging, validation of self-reported abstinence has become more common. Confirmations by informants in the participant's natural environment have been the mainstay (*8, 21, 22, 27, 28, 31, 32, 59, 64, 71, 85, 123, 141, 142, 197, 202, 206, 210, 229, 240, 242, 251, 279, 292, 313, 362, 394, 446*). However, carbon monoxide monitoring (*71, 206, 351*), threatened or actual urine nicotine analyses (*308, 409*), a bogus marketing survey procedure (*94*), and attempted (*80*) or actual (*48, 246*) thiocyanate analyses have now been reported. Although the outcome data on most procedures have been quite variable, the stricter methodology of these studies has encouraged continued refinement of interventions. More recently, effective multicomponent programs have begun to develop from this earlier research. The wealth of studies will be discussed briefly, therefore, with special emphasis given to those research trends that have produced programs with documented effectiveness. More detailed discussions of the literature are available in past (*24, 200, 230, 366*) and recent (*26, 29, 226, 245, 368, 376, 413*) reviews.

The research in this area can be grouped loosely into two broad, but not mutually exclusive, categories: (1) behavioral self-control strategies utilizing high participant involvement and (2) aversion strategies designed to reduce the probability of the smoking response (*226*). However, the most effective programs have tended to be multicomponent interventions which combine certain strategies from both categories.

Self-Control Strategies

Stimulus Control

The basic philosophy of behavioral self-control treatments has been to provide the subject first with increased awareness of the target behavior and controlling stimuli and then with specific self-management skills to control the target behavior (*13, 14, 193, 241, 314, 414, 415*). Therefore, self-monitoring of individual smoking behaviors has been a fundamental element in all behavioral self-control programs. As a sole treatment, self-monitoring has rarely produced more than temporary treatment effects (*60, 87, 109, 250, 251, 288, 365, 411*) and has been classed with the nonspecific treatment factors common to almost all behavioral programs (*251*). Self-monitoring has usually been combined within stimulus control treatments to make subjects aware of the specific environmental and internal cues associated with smoking urges and behaviors.

These stimulus control programs have been based on learning-theory formulations (*168, 169, 172*) of smoking behavior that suggested cessation is difficult because smoking is prompted by such a variety and range of cues. Subjects were taught to reduce the strength of these cues either by eliminating smoking from an increasing number of situations or by making time intervals the only controlling cue (*24, 26, 226*).

While this process theoretically should, with rare exceptions (*311, 344, 345*), make cessation easier, most subjects were reported to have difficulty reducing below 10 to 12 cigarettes per day (*8, 10, 23, 59, 104, 139, 221, 2*[illegible]*, 313, 377*). It has been suggested that, when most smokers reached that reduced level, each cigarette became more reinforcing and difficult to give up (*104, 243*).

Most studies involving a variety of stimulus control and other self-management techniques were shown to be at best only temporarily superior to control conditions. These studies have produced, in general, the common pattern of temporary reduction but rapid relapse and long-term abstinence rates that did not differ from those expected from nonspecific treatments (*10, 23, 60, 69, 87, 104, 125, 132, 139, 146, 155, 188, 191, 196, 197, 199, 221, 242, 260, 264, 273, 277, 279, 280, 328, 355, 365, 377, 385, 386, 411, 435*). Even when applied within more complex, multicomponent programs, the stimulus control-based treatments often produced only moderately encouraging findings (*48, 104, 155, 255, 273*). Some encouraging applications have been noted (*44, 45, 308, 416*), however, especially when the programs develop from systematic research and the programs offer behavioral training in a wide range of skills (*42, 310*).

Contingency Contracting

One specific technique that has produced some encouraging data involves the depositing of money for later disbursement based on attainment of specified goals. Early research on the technique was equivocal (*24, 200, 224, 230*), but several studies have produced impressive results. Elliot and Tighe (*95*) reported 84 percent abstinence at treatment termination, with 4 of 11 (36 percent) in two other groups followed up 15 to 17 months after treatment. However, the treatment also involved public pledges, stimulus control techniques, and group support.

Winett (*446*) found that 50 percent of the subjects in contingent repayment condition were abstinent, validated by informant reports, at 6-month follow-up, but only 23.5 percent of those in noncontingent repayment were abstinent. Multiple case studies by Axelrod and associates (*6*) and a study by Rovner (*342*) were also encouraging. Brengelmann (*44, 45*) has reported notable success in recent studies utilizing contingency contracting within a treatment-by-mail program. Forty-seven percent of those responding to the 15-month follow-up were reporting abstinence. However, self-reports were not validated, and if one assumed that nonresponders were smoking, the success rate based on all subjects completing treatment would be only 23 percent (22 of 96). Some success has been noted utilizing contingency contracting as a maintenance aid within a broad-spectrum program (*210*). In sum, as a single technique, contingency contracting appears able to initiate some behavioral changes, and when used in combination with other procedures, to prevent relapse.

Other Self-Control Strategies

Several other techniques or procedures have been modified for treatment of smoking behavior. Systematic desensitization was one procedure that was adapted for use with smokers under the rationale that reducing the need for stress-related cigarettes would aid subjects in coping with cessation. Again, while the technique was theoretically attractive, long-term abstinence rates were unimpressive (*96*, *200*, *205*, *215*, *263*, *301*, *426*). Similarly, a direct test of meditation proved to be equivocal (*287*).

In a similar vein, the suggestions of Homme (*163*) have produced a number of treatments attempting to increase self-control over smoking. Homme focused on "covert operants" which were designed to be incompatible with smoking behavior. He also reinforced nonsmoking alternatives. However, only temporary treatment effects were produced in control trials (*125*, *188*, *199*, *212*), despite some clinical demonstrations (*416*). Several other studies tried some combination of techniques along these lines with only minimal success (*38*, *120*, *281*).

Aversion Strategies

Techniques designed to reduce the probability of smoking through the use of aversive stimuli have been very commonly utilized in behavioral research projects. The theoretical underpinnings of individual procedures remain only partially delineated, and different theoretical positions—such as operant versus classical conditioning perspectives (*12*, *14*, *106*)—can result in varying treatment predictions (*26*, *226*). Possibly due in part to this lack of theoretical precision, early research on aversive strategies produced mixed results (*107*, *135*, *201*, *279*, *313*, *326*, *327*, *435*, *436*, *437*). Continuing refinements and evaluations have led to more elaborate combinations that appear more effective

Aversive control procedures can most easily be categorized according to the major stimuli used: electric shock, covert sensitization, or cigarette smoke. All but two studies (*242*, *434*) reporting minimal long-term results for taste aversion fit easily into these categories. The three major stimuli have rarely been used in combination with each other, but more recently have been included in multicomponent packages that include aversion and self-control strategies. For clarity, the research on the aversive control procedures applied in isolation will be examined first.

Electric Shock

Previous reviews (*24, 200, 230*) of early studies (*201, 279, 313, 435*) concluded that it was most likely that laboratory administered shock was ineffective because humans were too capable of discriminating between shock and no-shock situations. Thus, in spite of encouraging case study data (*338*), controlled experiments have failed to produce impressive long-term results (*20, 32, 64, 220, 350, 394*) or even superiority over attention-placebo controls (*20, 64, 350*). The nondifferential results from contingent and noncontingent shock conditions in the study by Russell and his collaborators (*350*) suggested that "traditional conditioning processes do not contribute significantly to the clinical response of human subjects to electric aversion therapy for cigarette smoking" (p. 103).

Some positive results are noteworthy, however. Berecz (*21, 22*) has presented interesting case study data suggesting that shocking imaginal urges rather than actual smoking may be more effective. Chapman and his colleagues (*58*) combined daily shock sessions with intensive self-management training to produce reported abstinence in 6 of 11 (54.5 percent) of the participants at a 12-month follow-up. Dericco, et al. (*85*) produced a clear treatment effect for electric shock therapy. Sixteen of twenty (80 percent) of the subjects receiving shock were abstinent at 6-month follow-ups with validation by informants. The treatment involved sessions 5 days per week for several weeks, with higher than normal shock intensities and the additive influence of other treatment factors. Thus, these results do not refute the basic conclusion of past reviewers that shock augmented by other procedures may produce an effective treatment package, although as a sole treatment it fails because the effects often do not generalize outside therapy (*200, 226, 230*).

Covert Sensitization

Cognitive processes have been commonly employed to produce aversion by pairing smoking with vivid images of extreme nausea or other unpleasant stimulation. This procedure of covert sensitization showed promise in case studies (*57, 416*), but experimental studies involving various types of control conditions or treatment comparisons have failed to produce either meaningful levels of long-term abstinence or superiority over controls (*14, 118, 212, 236, 249, 268, 280, 315, 355, 384, 426, 431, 447*). However, it has been suggested as a maintenance strategy (*29*), and variants of the technique have been utilized in the more elaborate multicomponent treatments to be discussed later.

Cigarette Smoke Aversion

The choice of cigarette smoke as the aversive stimulus in smoking treatment may be particularly appropriate because: (1) the reinforcing aspects of almost any stimulus are reduced if presented at sufficiently increased frequency or intensity, and (2) the aversion affects many of the endogenous cues that characterize smoking (*26*, *226*). Several main versions of this approach have been used: satiation (that is, doubling or tripling the daily consumption of cigarettes) prior to abstinence; and aversive conditioning through either smoking with warm, stale smoke blown into the face, or rapidly smoking with inhalations every 6 seconds.

Early research using artifically produced warm, stale smoke to affect aversion showed impressive initial results (*436*) followed by total failure during follow-up (*437*). Other early studies also produced minimal or no long-term successes (*107*, *135*). However, in a subsequent study with the warm, smoky air apparatus, Schmahl and his colleagues (*362*) produced both 100 percent termination abstinence and an impressive 57 percent (16 of 28) abstinence rate at 6-month follow-up, verified by random checks with informants. In the treatment, subjects were required to smoke rapidly (inhaling every 6 seconds) and continuously while facing into the blown smoke until further smoking could not be tolerated. Sessions were scheduled until the subject was abstinent a minimum of 24 hours and felt confident in maintaining abstinence (mean of about eight sessions).

A well controlled replication against a normal-paced, smoking attention-placebo control found 60 percent (18 of 30) abstinence among three experimental conditions at 6-month follow-ups, but only 30 percent (3 of 10) abstinence in the control (*229*); this was again verified by random checks of informants. As the rapid-smoking-only condition was as successful as the more involved procedures, abandonment of the inconvenient smoke blowing apparatus was recommended (*229*). Subsequent early research by Lichtenstein and his colleagues was also highly effective (*226*). The logic and supporting data for the procedure have been considered in more detail by Lichtenstein and Danaher (*226*).

Owing in part to the early effectiveness, convenience, and simplicity of the rapid smoking procedure, it became increasingly popular (*72*, *226*). Subsequent results are mixed and variable (*72*), however. A multiyear follow-up of the early studies has shown that some relapse did occur over the intervening years (*232*). Danaher (*72*) recently has comprehensively reviewed the existing data on the procedure and documented that termination and follow-up abstinence rates varied widely in subsequent research, with some studies reporting minimal or no (0 to 29 percent abstinence) long-term successes (*94*, *122*, *127*, *206*, *215*, *409*), others with moderate (30 to 49 percent abstinence) success (*28*, *31*, *104*, *202*, *207*, *209*, *276*, *292*, *325*, *452*), and a few approximately replicating the follow-up data of early studies (*71*, *94*, *144*, *246*).

Danaher (*72*) has attempted to clarify these data by highlighting the departures from original treatment procedures by the use of group presentation (*94, 127, 206, 209, 215, 246, 276, 292, 325, 352*), limiting the number of sessions (usually to six) (*123, 127, 202, 276, 292, 325*), offering treatment on a rigid or fixed schedule (*28, 71, 94, 123, 127, 202, 276, 292, 325, 409*), and omitting the contingently warm, supportive treatment context (*94, 206, 207, 209*). The most impressive recent outcome data have been produced with multicomponent approaches combining aversion and self-control procedures (*28, 31, 94, 144, 246*). Nevertheless, it is important to note that several multiple case studies and controlled studies on the rapid smoking procedure failed to demonstrate any improvement with the addition of self-control procedures (*70, 71, 123, 292*).

Thus, the rapid-smoking procedure appears to be a potentially very effective but complex intervention, dependent both upon the subject's active revivification of the aversion (*12, 226, 246*) and upon critical elements in the format, including a warm, personal client-therapist relationship offering social reinforcement and positive expectations (*72, 88, 226, 246*) and flexible or individualized treatment scheduling to insure total abstinence prior to treatment termination (*72, 226*). Numerous nonreplications and one direct test (*276*) have demonstrated that the production of only physiological aversion and conditioning effects are insufficient to produce long-term abstinence.

Satiation

Early research (*436, 437*) on the satiation technique was encouraging, with a 63-percent reported abstinence at 4-month follow-up. The success was partially replicated in a slightly modified, marathon format (*240*), but the weight of evidence on the procedure has been negative since that time. Controlled studies were unable to replicate the impressive cessation data or even to demonstrate superiority to control groups (*59, 211, 408*). Other comparative tests have also produced negative results (*32, 207, 242, 249, 280*). While the procedure as a sole treatment may have questionable effectiveness, more recent studies (*28, 31, 80, 210*), combining satiation with multicomponent treatment packages, have reported more impressive results.

Medical Risks of Aversive Smoking

Because the smoke-aversion procedures were developed to induce a degree of physiological discomfort by excessive smoking, the cardiopulmonary stress of increased nicotine and carbon monoxide exposure has been noted with concern, especially with regard to rapid smoking (*156, 164, 165, 223*). A number of studies have been undertaken to quantify the impact of rapid smoking on the cardiovascular system (*73, 78, 79, 144, 174, 261, 354*); much of the data has been summarized by

Lichtenstein and Glasgow (*228*). Recent studies by Hall and associates (*144*, *354*) and Miller and associates (*261*) have documented that the rapid smoking procedure produces an acute and dramatic effect upon vital signs (respiratory rate, heart rate, and blood pressure), blood gases, and COHb saturations, which make the procedure contraindicated for individuals with potential or active cardiovascular or pulmonary diseases. Adequate medical screening of potential treatment participants has been strongly recommended (*144*, *156*, *223*, *261*, *354*).

Data have yet to be published on the relative risks of other smoke-aversion procedures. If heavy-smoking subjects double or triple their daily smoking consumption during the satiation procedure, notable acute effects on the cardiovascular system may also occur. It should be noted that in excess of 35,000 participants have been exposed to the rapid-smoking procedures, with an informally reported morbidity rate from nonspecific complications of about 0.023 percent and no reported mortality (*228*). Yet, until the relative risks of procedures have been adequately researched, all the smoke aversion procedures should be used with appropriate screening and monitoring (*144*, *156*, *228*, *261*, *354*).

Less Stressful Alternatives

The identification of the relative risks of the rapid smoking procedure has stimulated the development of smoke aversion interventions that involve less physiological stress. Because of the pattern of 20 to 30 percent long-term abstinence with a common normal-paced attention-placebo condition (*71*, *123*, *202*, *206*, *207*, *209*, *211*, *229*), which self-control training seemed to enhance (*71*), initial clinical demonstrations have been undertaken combining normal-paced "focused" smoke aversion within broad, multicomponent treatment packages (*74*, *141*). Preliminary demonstration data showed that a 6-month abstinence could be produced in approximately 50 percent (5 of 10) of the participants (*141*). A controlled test of a rapid-puffing-sans-inhalation procedure produced somewhat less optimistic results with only 6 of 21 (29.6 percent) of the participants who started treatment reporting abstinence at the 3-month follow-up; this was verified by random checks of informants (*292*). A recent report by Tori (*417*) found that a smoke-induced taste-aversion technique involving limited smoke inhalation produced reported abstinence in 17 of 25 (68 percent) of the participants versus 6 of 10 (60 percent) in a rapid smoking condition at a 26-week follow-up. Unfortunately, assignment to treatment was not random, abstinence reports were not validated, subjects were treated on a fee basis, and a variety of adjuncts including hypnosis were utilized as maintenance boosters. Nevertheless, this and other early data (*74*, *141*, *292*) on alternatives to rapid smoking involving similar treatment formats, rationales, and nonspecifics, but markedly reduced physiological stress, appear encouraging and worthy of additional controlled research.

Multicomponent Interventions

As noted above, the research on techniques and procedures derived from learning theories and models has been mixed and often inconclusive. As recommended by early reviewers of the behavioral literature (*24, 366*), treatment packages combining multiple techniques are beginning to emerge. These comprehensive programs utilize some combination of the behavioral self-control techniques, and many also integrate aversive control procedures. The technology in this area is still developing; the early mixed results are to be expected. Still, recent reviews have uniformly concluded that the data from this emerging trend in programming are clearly encouraging (*26, 29, 226, 245*).

Treatment packages using behavioral self-control strategies alone have not produced notably effective results. Several complex programs have produced minimal long-term effects (*48, 104, 115, 255, 381, 382*). The later successes of Pomerleau and associates (*308*) and Brengelmann (*44, 45*) only came with refinements based on systematic developmental research. The most recent successful reports (*28, 31, 44, 45, 210, 246, 308*) thus appear to be a product of practical and in-depth knowledge of the problem which guides the application of the diverse elements in the treatment programs. Early and more recent successes (*28, 31, 39, 44, 45, 58, 80, 94, 140, 142, 210, 246, 308, 407*) suggest that planned extended contacts plus adaptation of techniques to individual needs are necessary for long-term success.

In a carefully evaluated clinical demonstration, Pomerleau and associates (*308*) reported success in 61 of the first 100 participants with 32 remaining abstinent (these were verified by urinary nicotine assays at 1-year post-treatment). Brengelmann (*42, 45*) has refined his complex treatment package (*42*) to the point where current results with treatment-by-mail are equal to face-to-face therapy, with 55 to 67 percent of the participants who complete treatment (86 percent reported completion rate) reporting abstinence at termination and 57 percent of those responding to follow-up reporting continued, but unverified, abstinence. Although the success rate based on the assumption that nonresponders were smoking would be 23 percent, the efficiency of the approach is clearly encouraging.

Other multicomponent treatments utilizing an aversion procedure to help induce cessation have also produced initially mixed but encouraging data. The early multiple case study of Chapman and associates (*58*) with electric shock plus extended self-management training is an often-cited example of this type of approach. In recent clinical evaluations of delivery formats, Best and associates (*28, 31*) have also documented the potential efficacy of a multicomponent program involving aversive smoking (satiation and rapid smoking) plus behavioral self-control training. Abstinence rates at 6 months, verified by informant reports, have varied from 35 to 55 percent, with the best results in a take-home version involving minimal personal contact. In a controlled study of satiation plus self-control training, Delahunt and Curran (*80*) demonstrated the superiority of the multicomponent treatment over controls and individual components. Six-month abstinence data showed five out of nine subjects (56 percent) for the combined treatment, but only 0 to 22 percent for individual components and controls; self-report validity was enhanced by collected but unanalyzed saliva for thiocyanate assays. Elliott's (*94*) package of rapid smoking, self-control strategies, covert sensitization, and systematic desensitization likewise produced abstinence, verified by a bogus marketing survey, in 45 percent (9 of 20) of the participants at 6-month follow-up, versus 17 percent for rapid smoking only and 12 percent for attention-placebo control. McAlister (*246*) demonstrated that his multicomponent rapid-smoking package was equally effective at 3-month follow-up presented either in person (56 percent or 5 of 9 abstinence) or over television (62.5 percent or 5 of 8 abstinence), with self-reports validated by thiocyanate assays.

These very positive findings are tempered somewhat by several less successful combinations of self-control and aversive smoking procedures (*27, 71, 123, 292*). The analytical study of the multicomponent approaches by Flaxman (*104*) provided some data on the complexity of the issues involved. Although the study indicated that subjects who abruptly quit on a selected date after self-control training reported the best 6-month abstinence data either with subsequent aversive smoking (5 of 8 or 62.5 percent) or only supportive counseling (4 of 8 or 50 percent), gradual reduction strategies, especially for male subjects, were markedly less effective with or without aversive smoking. Though the cell frequencies were small and the abstinence data unverified, the results suggest that successful response to multicomponent treatments may be the product of many only partially understood variables.

Treatment Innovations

Older (*371*) and more recent (*119*) survey data clearly indicate that most smokers who are motivated to quit are less interested in formal programs than in do-it-yourself methods. The broadening of the mode of service delivery of behavioral treatments is thus another encouraging trend. A study by Dubren (*90*) suggested that brief interventions by television can produce small but meaningful abstinence rates on the order of 9 to 10 percent. He also demonstrated that taped telephone messages can be used to extend the intervention and support maintenance (*91*). McAlister's (*246*) experimental demonstration of the potential of the media-only treatment group was impressive. Rosen and Lichtenstein (*339*) evaluated a program independently developed by the employer. They reported encouraging results using the resulting monetary contingency technique. These preliminary studies suggest that the best of the behavioral technology could be made available effectively by media or at the worksite to those smokers unwilling to attend formal programs.

The basics of successful clinical programs have also been reduced to self-study books (*310, 72a*). Consistent with the growing trend toward self-administered treatments (*124*), multicomponent treatments based on behavioral self-control strategies with or without aversive smoking techniques (*310, 72a*) are now available in self-study formats. Although initial tests of the self-study approach to smoking cessation are mixed (*28, 31, 123, 202*), their availability should facilitate further testing of programs similar to the successful self-managed clinic reported by Best and associates (*28, 31*).

Controlled Smoking

Most smokers want to reduce their risks from smoking (*49, 347*); this is evidenced by the dramatic changes that have occurred in the types of cigarettes being smoked (*151, 270, 287, 345*). Filter cigarettes are now the norm, and both the tar and nicotine content of the American cigarette have declined significantly (*279, 412*). These natural trends and apparent high interest among smokers in safer smoking have stimulated only preliminary interest in the development of interventions to maximize the reduction of risks (*49, 287, 347*). Frederiksen and associates (*108–112*), however, have pursued the topic and have experimentally demonstrated that exposure level can be controlled not only by rate of smoking and strength of cigarette, but also by altering the topography of the habit. They demonstrated that modifying the topography of smoking involves changing how much smoke is inhaled, how many puffs per cigarette are taken, and how much of each cigarette is smoked (*109, 110, 112*). Although the technology is still in the clinical-developmental stage, and the long-term stability of the changes will need to be verified, initial single-case demonstrations are encouraging and merit more emphasis. Data from the stimulus control studies suggest that reduction in exposure may be limited by the floor effect of 10 to 12 cigarettes per day (*8, 10, 23, 59, 104, 139, 221, 242, 313, 377*).

The controlled smoking technology may be useful to other groups of individuals. Physiological monitoring of ex-cigarette smokers who shift to pipes and cigars has documented that inhalation does occur (*81, 82, 351*). Because the inhalation may occur at an unconscious level and can lead to tobacco exposures as great as cigarette smoking, such smokers may need specific behavioral training to control the topography of their new habits. Similarly, some smokers who shift to lower tar and nicotine cigarettes to reduce their risk may also require the controlled

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smoking technology to avoid increases in rate or attempts to compensate by altering the smoking topography.

Maintenance of Nonsmoking

Both early (*24, 200, 366*) and more recent (*26, 29, 40, 226, 245, 306, 368, 376*) reviews of the smoking intervention literature have focused on the need to devote more energy to developing procedures to assure long-term, robust behavior change. The continuing problems of nonreplications and minimal treatment effects have, however, kept most researchers searching for new or more effective *cessation* strategies. Yet past research has clearly indicated that most smokers motivated to quit relapse shortly after treatment termination (*170, 171*). Thus all interventions should recognize that the production of the initial cessation is only the start of treatment (*26, 226, 245, 306*). Detailed procedures to aid the recent ex-smoker learn the skills needed to solidify the behavior change should become an integral part of all treatments.

Existing attempts to add maintenance programming to various treatments have proven somewhat ineffective (*306*). When offered booster sessions or telephone support if problems arise, most participants fail to make use of the services (*27, 380*). Experimental tests of the booster treatment approach generally have shown equivocal results (*84, 202, 325*). Paradoxically, supportive phone calls during or after treatment seem to lead to significantly poorer long-term results (*28, 84, 380*). It has been suggested that maintenance programming must be offered in a fashion that will enhance rather than distract from self-attributions of success (*29, 203*).

Some initial positive findings are available, however. Dubren (*90*) reported some success utilizing tape-recorded telephone reinforcement messages during the follow-up of a televised smoking clinic. After some initial negative and inconsistent results (*206*), Lando (*210*) demonstrated, but was unable to replicate, that the long-term effectiveness of an aversive smoking program may be enhanced by a broad-spectrum, contingency-contracting program. Seven maintenance sessions over a 2-month period produced abstinence, validated by informant reports, in 76 percent (13 of 17) of the maintenance group subjects at 6-month follow-up, versus only 35 percent (6 of 17) of the controls given cessation treatment only. Case study data support the maintenance-contracting concept (*222*). Recent dissertation data also appear to provide some encouraging findings regarding maintenance programming (*84*).

Attempts to add on maintenance procedures have generally been ineffective (*27, 31, 202, 206, 292, 356*). However, several effective programs appear to have integrated into the total treatment package extended contacts and training in the behavioral skills (*28, 44, 45, 58, 210, 308*). These factors may be required to maintain abstinence. More research is needed to define what types of maintenance procedures are needed and when and how they can be most effectively administered (*306*).

Research has begun to clarify the personal and situational factors which support smoking and which may induce ex-smokers back into the habit (*30, 97, 110, 111, 243, 256, 349, 359*). Individual difference factors have been overemphasized in the analysis of relapse, however, compared to situational factors (*29*). Retrospective analyses of individual differences that may be related to successful cessation have generally suggested that older males with lighter smoking habits and from higher social classes tend to be more successful (*92, 126, 149, 233, 271, 323, 389, 390*), but the magnitude of these differences has been small (*29*). Several studies have suggested that individuals who report using smoking to control negative affect or who have higher levels of anxiety also appear more susceptible to relapse (*89, 105, 179, 180, 292, 370, 375, 389, 390, 399, 400*). Efforts to utilize broad individual differences to maximize treatment effectiveness have been mixed and generally inconclusive (*27, 32, 33, 53, 205, 212, 292*). Given that broad smoking topographies (*1, 29, 176, 177, 256, 349*) and personality tests (*27, 179*) lack sufficient specificity, Best and Bloch (*29*) have suggested that emphasis should be placed on locating interactions between finer variations in the individual's situational cues and smoking patterns (*30, 97, 110, 111, 243*) and responsiveness to treatment modalities.

McAlister (*245, 246*) has outlined several other important areas that should be addressed in maintenance programming. Smokers need to be given a positive set regarding withdrawal symptoms and their ability to deal with them. Some data suggest that misattribution-type therapy can be helpful in achieving this goal (*16, 245*). Since most smokers, especially women, believe they will gain weight if they quit (*271*), fear of the documented weight gain after cessation (*37, 50, 62, 122*) should be directly countered (*245*). The role of negative self-evaluations and common rationalizations (*76*) also requires further clarification (*13, 245*). McAlister (*245*) has suggested that specific plans be formulated to aid ex-smokers confront their predicted problem areas.

Research interest in the important area of maintenance programming is beginning, but many issues remain to be defined and tested. Preliminary data suggest that multicomponent programs are more effective when extended contacts are planned into the program and diverse techniques are individualized to meet the special needs of all participants. Given the concern over smoking among women (*65, 162, 214, 335*), their special needs should be addressed.

General Overview of Data

Status of Methodology

As stated at the beginning of this section, there have been great improvements in the quality of data on smoking cessation methods in recent years (*26, 226, 368, 376*), especially in several research clinics (*81, 82, 178, 283, 381, 382*), large-scale coronary prevention trials (*101, 265, 266, 324, 441*), and in the behavioral research area (*26, 29, 226*). Yet the validity of the self-report data remains a critical concern. Since the validity of reported abstinence has been questioned by physiological measures in up to 20 percent of clinic participants (*47, 82, 178, 231*), it appears that many individuals may be reporting their commitment and expectations of success rather than their current smoking behavior. Ohlin and associates (*283*) revealed that, of the 19.2 percent (25 of 130) of the reportedly abstinent subjects who had COHb levels above a 0.8 percent nonsmoking cutoff at treatment termination, none was reporting abstinence at 6-month follow-up. With the current state of unverified self-report data, one must interpret cautiously even the commonly cited relapse curves (*170, 171*).

Random assignment to experimental conditions and the use of one or more control conditions have become much more common, especially in the behavioral research areas. Broad generalizations of the data continue to be made about the general efficacy of procedures with little regard for the interactive effects of age, gender, social class, or smoking topographies of successful participants. The small samples of almost all comparative research relegate these sources of possible interaction to the error variance. This, plus wide variability in the actual application of supposedly identical procedures, makes comparisons across individual studies difficult.

The continuing pattern of nonreplication and the lack of clear superiority of treatments over appropriate controls further suggest the need to balance these advances in research methodology with a practical and clinical sensitivity to the complexity of the problem (*7, 43, 224, 225, 304*). The guidelines offered by several comprehensive clinics (*43, 224, 304, 372, 375, 379, 380, 381, 383, 440*) should serve to direct initial clinical testing of procedures. As McAlister (*245*) has outlined, procedures should first be intensively piloted with single individuals or small groups. The technology for the use of quasi-experimental (*56, 393*) with other methods should make it possible to conduct multiple case studies with adequate statistical validity (*108, 158a, 293, 415*). When clinically refined, the treatment techniques can be tested against appropriate controls, especially attention-placebo controls (*24, 56, 226, 251, 272*). When the format and techniques are well understood and documented, they can be replicated by other researchers in diverse settings (*245, 304, 398*).

Although behavioral research has been advancing in experimental rigor, less progress has been made in public service and proprietary clinics. Objective and controlled evaluations are still needed in these settings. Though the treatment focus of these clinics makes classical experimental designs unattractive, alternative quasi-experimental designs should be investigated, since the technology exists to provide a degree of control in almost any field or applied setting (*56, 393*). If such evaluations were undertaken, a wealth of data would be available to guide more controlled research (*398*).

Most researchers now seem at least aware of the need to conduct long-term follow-ups of all participants. While various professional and financial constraints tend to limit this process, follow-ups of at least 6 months are becoming common. Innovative suggestions, such as obtaining the name of a contact who will know the future whereabouts of the participant, have been offered to aid in tracking participants during follow-up (*232*). The public service and proprietary clinics are only beginning to recognize their responsibility in this area, and little is known about the long-term efficacy of these programs.

In summary, the research on smoking-modification strategies over the past 15 years clearly indicates that past recommendations regarding adequate methodology still need to be heeded (*24, 26, 226, 251, 272, 366, 376*). Researchers also need to become more aware of social contingencies such as clinical zeal, publication pressures, and dissertation timetables which have led to poor adherence to these guidelines (*225*). Data on the reliability and validity of self-reports of smoking behavior now strongly suggest that unverified, global self-reports should no longer be accepted as the only outcome data. Objective techniques for measuring smoking exposure can be developed to validate and supplement self-report data. While great advances in methodology have been made in the past 15 years (*26, 226, 376*), new technical and design approaches now under study should serve to improve further the quality of the data collected in the future.

Implications of the Data

In light of the amount of research conducted over the past 15 years, it is remarkable that we have so little outcome data on the wide variety of treatments being offered and recommended. Equally astounding is how little we know about the millions of smokers who have quit on their own. As noted in other sections, it has been estimated that 95 percent of the 29 million smokers who have quit since 1964 have done so on their own (*270*). Various surveys have revealed that the cumulative quit rates for various age groups, social classes, and occupations are impressive (*92, 121, 133, 149, 271, 323, 421*). The sporadic and marginal quality of outcome data on treatment programs, however, makes it impossible to conclude how this broad social phenomenon has affected clinical and research programs. Survey data

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have shown that only a third or less of smokers motivated to quit are interested in formal programs (*119, 371*), and only a small minority of those who do express an interest actually attend programs when they are offered (*195, 270*). It thus appears that objective outcome data that are available may be based on a small minority sample of smokers at large.

Objective data are lacking on most of the smokers who have been willing to attend formal programs. Public service clinics continue, but the lack of objective outcome data precludes the evaluation of their efficacy. Similarly, proprietary programs remain virtually unmonitored and unevaluated in an objective fashion. Smoking counseling by medical or health care personnel seems to be highly effective with symptomatic smokers (*227, 338*), but the efficacy of such an approach for other smokers has yet to be adequately evaluated. The data from the large scale coronary prevention trials (*101, 265, 266, 324, 441*) should help clarify some issues regarding medical counseling and smoking cessation among higher risk individuals, but the nonspecific treatment focus of these projects will limit the conclusions that can be drawn.

Controlled research has yet to produce a clearly superior intervention strategy. However, the rapidly accumulating and improving research data now suggest that multicomponent interventions offered by intervention teams with practical knowledge regarding the smoking problem are the most encouraging. In part, the added effectiveness of some programs may be due to the skills of the intervention team to present the available techniques as both credible and attractive to the participants (*173, 175*). It is important to recognize that improved success in recent studies may also be influenced by changes in social norms regarding smoking. More integration of diverse perspectives, including pharmacological, behavioral, medical, and social aspects of the smoking habit, should enhance the multicomponent treatment approach. It is encouraging to note that more research emphasis has begun to be focused on maintenance programming. Apparently the multicomponent programs enable participants to gain the new skills needed to deal with their individual problems in adjusting to the new nonsmoking lifestyle. Many issues remain to be researched, however, and special programs may be required to deal with the needs of smokers with personal or environmental factors that encourage recidivism.

Recommendations for Future Research

Objective Measures of Smoking

An adequate technology to validate self-report smoking data is critically needed. When physiological assessments have been done, inaccuracies in self-reported abstinence are common. Inaccuracies in rate estimates among the continuing smokers cannot, however, be accurately evaluated with existing technology. If reliable physiological measures of smoking rate were available, the effects of various procedures in producing not only abstinence but meaningful and enduring reductions in smoke exposure could be objectively verified. Basic pharmacological and biological research is needed to formulate such objective measures of smoking.

Maximizing Unaided Cessation

The phenomenon of smoking cessation outside formal programs remains largely unexplored. Almost all successful ex-smokers quit on their own, but little is known about how to maximize this process. Existing survey data suggest that most smokers who are motivated to quit are not interested in attending formal programs. Most smokers report being interested in do-it-yourself quit methods or procedures. Therefore, precise information is needed regarding what types of treatments smokers view as credible, useful, and attractive. Controlled research is needed to evaluate the most cost-effective programs to make attractive and effective programs available to smokers who desire to quit. As treatments are refined in controlled research, they need to be translated into formats which are appropriate for testing with general population groups.

Development of Maintenance Strategies

The research on methods to assure that smokers who successfully quit have the behavioral skills and social supports needed to maintain and solidify the behavior change is currently at a very primitive stage. More basic research is needed to clarify the topography of smoking and relapse behavior so that the specific needs of various types of smokers can be fulfilled. Procedures and programs to aid smokers achieve cessation must be refined; past experience shows that the production of high rates of initial abstinence does not insure a noteworthy level of long-term abstinence. Different classes and types of smokers may require different levels of maintenance assistance. Specific smoking topography variables that predict such needs should be defined. Existing research on maintenance programming indicates that the maintenance procedures should be integrated into the treatment package rather than added on as an option at the end of the treatment. The development of maintenance strategies should be viewed as an integral part of the intervention package and should be evaluated accordingly.

Evaluation of Existing Programs and Procedures

As should be clear from the review of existing data, methodologically sound evaluations of all forms of smoking intervention are still greatly needed. The increased rigor in the behavioral research area has begun to produce some tentative suggestions regarding effective strategies. However, the more promising multicomponent treatment packages pose new, more complex issues for evaluation. Alternative methods of effectively presenting the most effectual programs to the general public need to be explored and properly evaluated. In addition, the most attractive of the behavioral programs should be experimentally tested relative to other existing intervention strategies in order to produce relative outcome data for evaluation.

The potential efficacy of smoking cessation and reduction counseling by physicians and health care professionals also should be experimentally evaluated. The existing technology derived from behavioral and social psychological research should be integrated into interventions appropriate for use in medical settings.

All public service clinics and proprietary programs should be subjected to rigorous and continuing evaluation. Such programs must recognize their responsibility to the smoking public to present objective evaluations of long-term effectiveness. In addition, proper evaluations should lead to refinements in treatment procedures. As effective treatment strategies are developed and objectively evaluated within research programs, they should be translated into clinic formats for utilization and evaluation within the general population.

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